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ON INSOMNIA AND OTHER TROUBLES CONNECTED WITH SLEEP IN PERSONS OF GOUTY DISPOSITION.

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THE sleeplessness which affects persons who are goutily disposed has not, so far as I know, received the attention which it demands. The subject has not been systematically treated by any author with which I am acquainted. Stray allusions have been made to it. There is perhaps not very much to be said about it, but it is fitting that such knowledge as is possessed should be carefully collated and recorded.

In this communication I propose to set forth all that I know about the matter, merely prefacing my observations with the remark that in this endeavour I shall strive to treat the subject from a simple clinical standpoint.

In an essay published in 'BRAIN' in April 1880, I alluded to the fact that there was a "well-marked variety of insomnia" in gouty persons. That this form of sleeplessness is well-marked, and is likewise peculiar, I shall try to show.

In the first place, it may be remarked, that I do not allude to the interruption of sleep which is caused by the sudden onset of an acute attack of gouty inflammation, and which so commonly occurs in the early morning hours. This is an extreme, and very exquisite, instance; albeit, the special phenomena associated with such an attack do indeed, I believe,

afford a type upon which the less-marked and minor symptoms of gouty insomnia are founded.

The simplest form of insomnia which occurs in persons who are goutily disposed is due in most instances, I believe, to dyspepsia. The earliest and best account of this with which I am acquainted, was given by Cullen,[1] who remarked that "persons who labour under a weakness of the stomach, as I have done for a great number of years past, know that certain foods, without their being conscious of it, prevent their sleeping. So, I have been awaked a hundred times at two o'clock in the morning, when I did not feel any particular impression; but I knew that I had been awakened by an irregular operation in that organ, and I have then recollected what I took at dinner which was the cause of it." This sleeplessness is often caused by some particular article of diet which the sufferer digests imperfectly, or may be due to excess of wine, or mixing of various liquors. Very often fatty or saccharine matters in excess, or mixture of fruit and wine, may cause this dyspepsia. There may be no overt symptoms of dyspepsia, but a simple excess in eating, or a single article of diet which is digested with difficulty, may so disturb the cerebral circulation that sleep is interrupted and suspended for a time. I described these symptoms in a paper which was published in 1873, and stated my belief that they were more common in persons who had tendency to gout.[2]

The dyspepsia probably arises from faults not only in the stomach, but in the duodenum and upper part of the small intestine.

Dr. Murchison has described this form of sleeplessness, and attributes it to the hepatic derangement which induces lithæmia and other symptoms of gout.[3] A very noteworthy point about such cases is the particular time at which the insomnia begins. The sufferer retires to rest feeling quite well, and free from any discomfort. But his sleep is rudely interrupted, it may

[1] Works, edited by Thomson, vol. i. p. 127. Edinb. 1827. Cullen did not, himself, connect this symptom with gouty tendency.

[2] On certain forms of sleeplessness. Brit. Med. Journal, Dec. 27, 1873, and republished by Longmans, 1874.

[3] Lect. on Dis. of Liver; edit. 2, p. 590. Lond. 1877.

be by some unpleasant dream, and he is at once aware of uneasiness in the stomach, has heartburn, or flatulence, and perhaps nausea.

If nothing be done, the patient will lie awake, with throbbing head and active flow of thoughts, for an hour or two, when sleep will return. On rising the next morning, he will probably experience some headache, and find his appetite diminished. An attack of hemicrania may perhaps render the next day miserable for him.

Relief may, in general, be readily found by taking a well-diluted dose of some alkaline salt, or by means of a drachm of compound rhubarb powder. If vomiting can be induced, great relief will follow, and a copious draught of cold water may be taken. Sleep will soon return, and no discomfort will be experienced the following day.

Such a form of dyspepsia is plainly a manifestation of a gouty tendency. This incapacity to digest certain definite articles of food is very marked in the gouty, and it is not unfrequently one of the earliest tokens of the disorder.

In youth there may be vigorous digestion for all kinds of food, but as the third decade is approached, the inability declares itself.[1]

The prevention of such disturbance is naturally secured by attention to diet, and omitting each article of food which is found to disagree.

It might be objected that there is nothing very remarkable about such symptoms, and that they are common enough. The characteristic part of the disturbance is the special digestive inadequacy at a very definite period. If we suppose that this enfeeblement is due to the taking of a full meal late in the day, and that the digestive powers would be adequate to dispose comfortably of the same if taken early, which is fairly conceivable, we have no means of knowing whether sleep, were it sought after some hours, would be interrupted under such circumstances. The fact remains that in persons of gouty constitution, sleep is apt to be disturbed by the

[1] This subject has been discussed by me in (1) "Studies of some Irregular Manifestations of Gout." St. Barth. Hosp. Reports, vol. xv. p. 105; and (2) "On Unequivocal Gouty Disease." St. Barth. Hosp. Reports, vol. xvi. p. 185.

irritation arising from their peculiar digestive incapacity, and at a definite period of that process. An interval of four or five hours occurs between the meal and the awaking, the patient being disturbed within about two hours of retiring to bed. This is the time at which attacks of gout are especially liable to come on, and the sufferer is suddenly awaked with pain in the affected part. In this case, as usually in that of gouty dyspeptic insomnia, the patient has retired to bed feeling comfortable, and in his ordinary health.

Other forms of gouty trouble manifest themselves not uncommonly in the early hours of the morning. Thus, attacks of bronchitis, with asthmatic dyspnœa, sometimes replace, or alternate with, regular onsets of gout, and the paroxysms of asthma are very prone to begin, and to disturb the patient, after midnight.

Not only is sleep thus interrupted, but other peculiar symptoms are met with in those of gouty proclivity in connection with the sleeping hours. It has been observed that in cases where a regular attack is expected, but does not supervene, sleep is abruptly broken some hours before the usual time of waking, and does not return. Some horrible dream may lead to this, and the same occurrence may take place for several mornings in succession. Scudamore relates two cases where sleep was merely disturbed by uneasy dreams, and gout was established in the joints on awaking in the morning.

Startings and shouting have been noted, associated, or not, with the dyspepsia preceding or accompanying gout. Severe and troublesome priapism in the night sometimes greatly annoys the gouty, commonly without erotic feelings. Grinding of the teeth during sleep is a symptom met with in those who are gouty. Dr. Graves first called attention to this. Dr. Garrod has no experience of it. I have collected several examples of it, and Dr. Donkin likewise directed attention to some well-marked cases which occurred in the family of parents who were both gouty.[1] In this family there was also history of somnambulism. The father, a gouty man, was a habitual somnambulist in early life, and occasionally in later years

[1] 'Brit. Med. Journal,' Feb. 21, 1880, p. 279.

walked about in his sleep. The mother ground her teeth at night for many years. She was of gouty parentage, as already stated, but had had no overt gout herself. The whole family, of eight children, ground their teeth almost incessantly at night. Most of them were extremely "nervous," and walked in their sleep. They also talked during sleep.

I have record of one case in which there is both tooth-grinding and occasional somnambulism, the mother and maternal grandfather being distinctly gouty. Nightmare and startings of the limbs have been observed with some frequency in persons goutily disposed.

In connection with the subject of insomnia in the gouty, may be noted the fact that many of the special determinations of the malady take place during the night, whether the sleep be disturbed or not. The patient retires to bed feeling in his usual health, but on awaking in the morning he discovers at once some new phase of his malady; it may be muscular pain or stiffness, angina of the fauces, the beginning of a hemicrania, or more or less severe pain in some joint or adjacent texture, such as a stiff-neck, lumbago, or a burning phalangeal joint. These troubles, or some of them, have come on in the night, but have not been sufficient to disturb sleep. Cramps in the calves of the legs are especially prone to vex gouty persons at night, and sometimes precede a severe attack for several nights.

The fact that not only acute attacks of gout are apt to supervene during the hours allotted to sleep, but that other less severe gouty manifestations likewise occur during the night, or are found to have come on at that period, is one amongst many which may be appealed to in proof of the neurotic theory of this malady, for it has this peculiarity in common with several other morbid affections which are conceded on all hands to be distinct neuroses. Thus, epilepsy, neuralgia, spasmodic asthma, gastralgia, angina pectoris, laryngismus stridulus, and hemicrania are all prone to disturb sufferers during the early hours of sleep, or immediately on awaking. In all these cases we have to seek for a cause which determines these outbreaks with such marked constancy in connection with the sleeping state.

From the nature of the case, we have but scant knowledge of most of the physiological conditions which occur during sleep. It is, however, known that the bodily temperature falls both in health, and in most, if not in all, morbid states, between the hours of midnight and six o'clock in the morning. Some observers have noted the minimum temperature to occur between 11 P.M. and 3 A.M., and although the fall does not amount to more than one or one and a half degrees, it has nevertheless a distinct significance as indicating some direct nervous influence on heat-production. Certainly, this constant and normal reduction of temperature is independent of removal of clothing, and abstraction of heat by bedclothes. Again, the subjective sensation of chilliness about three o'clock in the morning is familiar to all who sit up at night, and at that period, too, there is a maximum of weariness and exhaustion, and the greatest instinctive demand for sleep. Even the worst sleepers will commonly fall asleep at this hour, although they may have been miserably wakeful and restless previously.

There is also a greater susceptibility to cold at night-time. The "middle watch" is the most trying in all respects. This is the period of the greatest exhaustion of the whole nervous system, the automatic cerebral activity ceases, and sleep is "the diastole of the cerebral beat."[1] As the old writers put it, the "brain-power is lowered" in sleep.

Digestion is feebler during sleep than in the waking state, and so, too, is the action of the heart, and both the circulatory and respiratory acts are reduced in force and frequency. With the exception of the cutaneous functions, perhaps, all others are at rest as far as possible.

Sleep is more profound in the earlier hours of night, and gradually becomes less so towards morning.

It seems impossible, in view of the foregoing considerations, not to find some reasons for the marked tendency towards irregular outbreak of nervous energy during the hours when so many cyclical processes are modified or interrupted.

In perfect health, and in persons not neurotically disposed, no irregular effects ensue, but in morbid states, and in the neuroses, the hours of sleep are particularly those in which we

[1] Foster, 'Text Book of Physiology,' p. 573.

might expect some outbreak or irregularity, and, as a matter of experience, we find such to be the case in marked degree.

The anæmia of the brain in sleep may have some influence in determining some of these disorders; but this condition is not believed to be the cause of sleep, but only an effect or concomitant of it. In the cases of dyspepsia already considered, there is a manifest source of irritation at a distance; but the peculiarity here is that it only becomes potent at a definite time to disturb sleep, either by the generation of some special morbid product in the course of digestion, which may act from a distance reflexly, or may enter the circulation and rouse the higher centres. These centres may, during their temporary depression, due to sleep, be more than at other periods specially irritable, and in the cases of such persons as are neurotically disposed, they are almost certainly in less stable condition than are those of the healthy.

The direct influence of an excess of uric acid circulating in the blood can hardly be lost sight of in connection with gouty insomnia. It is well recognised that such excess is frequently present without the induction of any overt disturbance, nervous or otherwise, in persons who have no gouty proclivity; but in any case of true gouty habit we must not ignore, as it is perhaps now too much the fashion to do, the influence of what, when in excess, is a real poison in the circulation.

The special determinations of gout to certain parts are inseparably connected with excess of uric acid in such parts, and thus we may fairly conceive that some of the nervous symptoms which occur in the gouty owe their cause to irritation of nervous tissue by this peccant matter. Insomnia may well be one symptom due to this irritation.

It is to be noted that these troubles in connection with the sleeping state are not only met with when the subjects of them are in a very gouty condition, or as precursors of overt outbursts of gout in the arthritic form; they form part of the many minor affections to which persons goutily disposed are sometimes liable. Gout, like other maladies, has varying significance in the particular individual affected, and the fact of goutiness so far modifies the constitution or bodily habit of the patient. Thus, many sufferers have no troubles connected

with the sleeping state, just as many have no urinary difficulties, no hemicrania, and no tophi. Many attacks of gout set in violently by day, and not by night. These facts in no degree minimise the value and importance of accurate observations on the symptoms presented during sleep in persons disposed to gout. We must not fail to recognise their special significance when we meet with them.

Many sufferers are good sleepers in the intervals between severe attacks, and many others can secure good nights with due precautions as to diet and other habits. The particular insomnia described is rather the indication of the gouty habit, than a particular phase of either acute or chronic gout, as commonly understood, and, as such, its importance has not hitherto been clearly signalised. Its recognition is necessary for the employment of the only line of treatment that can truly avail to avert it, and to break the persistence of the habit on which it depends.

Hypnotic agents constitute improper means of relief in such cases as I have described. The gouty taint must be combated. Strict attention to diet, a free dilution of the blood with bland fluids, regulated exercise of both mental and bodily faculties, together with occasional mercurial purgatives, will commonly avail to overcome the misdirected tendencies, and to secure good nights for sufferers from these unpleasant affections.

ON ALLOCHIRIA.

A PECULIAR SENSORY DISORDER.

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THOUGH great progress has been made of late in the methods of investigation of diseases of the nervous system, yet there is an affection of sensibility which has hitherto escaped special notice. It consists in the fact that though the sensibility is retained more or less completely, yet the patient is not clear, or is frequently, if not constantly, in error, as to which side of the body has been touched. I would term the phenomenon "Sensory Allochiria," or shortly "Allochiria" (ἄλλος, χείρ) or confusion of sides. This symptom is so easy to determine that no long description of method is required. When we test the sensibility of a patient, on the leg for example, and find that the two points of the æsthesiometer are recognised as such at a moderate distance apart, or when a prick or pressure is fairly localised, and yet, as frequently occurs, the irritation is referred to the other side, we have the condition before us. The power of localisation is retained as to details, while doubt or error exists as to the side touched, the irritation being commonly referred to the corresponding part of the other limb. I believe that the phenomenon is less rare than overlooked, as errors on the part of the patient are apt to be regarded as merely a *lapsus verbi*. In the case of intelligent patients, however, and with due care, it soon becomes apparent that this is not the true explanation. First let me give a few illustrations.

CASE I.—A chemist, ætat. 35, who had had syphilis, and had been exposed to extreme cold, began soon after to suffer with the symptoms of slowly progressive sclerosis of the

posterior columns. After eight years he fell into a state of profound melancholy which lasted for a year. On his recovery from this condition I tested his sensibility. At this time he was able to walk a few steps alone, with his eyes open. Only the essential points need be mentioned.

The patient had a good, if not completely normal, judgment as to the position of his lower extremities, which were freely movable. Heat and cold were readily distinguished in the feet. Simple touch or gentle pressure were not perceived. Investigation with the æsthesiometer showed on the right indistinct diminution of the power of localisation, but not to any marked extent. A double impression was distinctly perceived at the following distances :—

Ball of the toe	R = 20mm	..	L = 12mm
Under the little toe	R = 17mm	..	L = 12mm
Middle of the sole	R = 9mm	..	L = 9mm
Dorsal surface	R = 12mm	..	L = 8mm
Inner side of the leg	R = 10mm	..	L = 7mm

The points required to be pressed deeply in order to elicit distinct sensation. The sensation was markedly retarded. As a rule the point touched was correctly given, only in a few instances an error was made, the patient believing the sole was the part touched, whereas the points of the æsthesiometer were on the toes. Frequently the impression was referred to the other foot, but localised in the correct position, to the astonishment of the patient when he was informed of the error. The patient continued under my observation six months after recovery of his mental equilibrium. He died three years after.

Case II.—W., a labourer, on March 25, 1878, received a severe blow on the back from a beam and was knocked down, paraplegic both as to sensation and motion, but without loss of consciousness. I saw him in December 1878. At that time he had some power of movement of the thighs, but was completely paralysed in the legs and feet. As regards sensibility, the patient made no complaint. He could distinguish hot and cold. On the inner side of the plantar aspect he could distinguish two points at a distance of 9–10 mm.

But he was frequently wrong as to the exact locality of the impressions in the limb, and was seldom right as to the side actually touched. The leg and foot were swollen, the feet turned inwards. On the sacrum there was a slight bedsore; previously the heels had been similarly affected. The urine was not passed involuntarily, but could not be retained long. As to the later progress of the case, the following notes were supplied me by my friend Dr. Pfleger.

During the month of June 1879, considerable œdema occurred in the lower extremities, and sores on the toes. The patient complained of formication in both legs, and soon after febrile symptoms occurred, with erysipelatous redness and swelling of the legs, with considerable diminution of sensibility. These symptoms subsided in a few days, and the sensibility reached its former grade. In October 1879, there were spasms in the thighs, and pains shooting from the hips to the knees. Later the decubitus in the sacral region extended and deepened, and the patient died in a state of marasmus on February 5, 1880. The autopsy was performed by Dr. Weichselbaum.

The body was of medium build, muscular; the lower extremities were œdematous. There was a large decubitus on the sacrum, the size of a small plate. The spinous process of the third lumbar vertebra projected abnormally, and the bodies of the first, second, and third lumbar vertebræ were anchylosed. The anchylosed region was hollow anteriorly, projecting with its convexity into the vertebral canal. The intervertebral discs were only partially retained in this region. The inner surface of the dura mater in the region of the *conus medullaris* and upper extremity of the *cauda equina* was covered here and there with a soft, rusty-brown pseudo-membrane, and united at these spots firmly to the arachnoid. The posterior surface of the *conus medullaris* was flattened, the substance of the cord itself appeared for an extent of 2 cm. of a yellow colour, shining through the arachnoid and pia mater. The lumbar enlargement was not distinct. On transverse section it was seen that in the region of the *conus medullaris* the substance of the cord, with the exception of the anterior columns, and the anterior position of the lateral columns, was converted into

a soft, yellow-brownish mass, strewed with whitish points and lines.

Higher upwards, towards the medulla oblongata, the median portions of the posterior columns was converted into a yellow-brownish wedge-shaped strand, traversed by a few whitish lines. The examination of the cord, hardened in bichromate of potash, was made six months after.

The substance of the cord, from the *filum terminale* to the upper third of the lumbar enlargement, felt spongy and unfit for section. Grey and white matter were undistinguishable. Teased specimens showed that the lowest part of the cord, including the lower third of the lumbar enlargement, was almost entirely degenerated. Besides a finely granular débris there were large numbers of fat granules and nucleated fatty cells. As remnants of the ganglion cells were certain multiform conglomerations, they were without nucleus, but contained one or more vacuoles, and became deeply coloured with carmine. The vessels which traversed the mass were covered on their exterior with a thick layer of fat granules. The connective tissue septa were thickened and contained the mass of detritus in their meshes.

In the upper portion of the lumbar enlargement the anterior horns were distinctly visible, and at the commencement of the dorsal region the whole aspect of the grey matter was beautifully distinct. Fine sections in this region exhibited well-marked ascending degeneration, which occupied the whole of the posterior columns. Only very few normal nerve fibres (9–10) were to be found in the most lateral portions, close to the posterior horns. On both sides also, at the periphery of the lateral columns close to the most external of the anterior root-bundles, there was a region visible to the naked eye by its lighter colour. Whether these symmetrical spots indicated ascending degeneration I have not been able to determine. There was visible also, throughout the whole end up to the medulla oblongata, an increase of the neuroglia, most marked at the periphery. This increase was particularly marked at the points in the lateral columns indicated, but it was not so far advanced as in the posterior columns. These regions were entirely distinct from Flechsig's ascending cerebellar tracts.

In the posterior columns the larger vessels, in consequence of the multitude of fat-granular cells on their adventation, appeared even to the naked eye as whitish cords. Successive transverse sections of the cord from below upwards showed that the degeneration for a length of 3 cm. occupied the whole breadth of the posterior columns, and gradually retreated from the sides towards the middle line, the line of separation from the normal lateral tracts being however at first indistinct. Certainly the degeneration extended quite up to the grey commissure. In the cervical enlargement the degeneration was confined to the columns of Goll, forming a sharply defined wedge-shaped area. The point of the wedge did not however extend as far as the grey commissure. There was no degeneration of the direct cerebellar tracts, owing to the lesion being situated so low down. But in most sections of the cervical enlargement there was a peculiar alteration of the grey substance. On both sides of the cord in front of the head of the posterior horns where they are narrowest, the grey matter was transversely divided by a peculiar, structureless, transparent mass intensely coloured by carmine, and very similar to the mass which is found round the larger vessels in inflammatory processes in the cord (Myelitis, Lyssa, &c.). I do not take upon me to attribute the mass to the same origin, but it seems worthy of note that such an exudation should occur in a region not particularly rich in blood vessels.

In the highest sections of the cord, the ascending degeneration of Goll's columns was beautifully distinct. The ganglion of the *funiculus gracilis* appeared also to have suffered degeneration. Along with fatty granular cells and connective tissue corpuscles, there was a considerable increase in the neuroglia, and the processes of the ganglion cells were fewer than in normal conditions. Yet there were normal fibres, which towards the upper extremity increased in number with the cessation of the degenerative process. Already in the upper cervical regions the degeneration again reached as far forward as the grey commissure, and in the region of the *calamus scriptorius* the sclerotic process seemed to extend forward between the nuclei of the hypoglossal nerves. The degeneration could not be traced further upwards.

Case III.—Dr. N. Weiss kindly showed me a patient who exhibited the symptoms of allochiria, but this was dependent on the nature of the irritation. The case was an ordinary one of sclerosis of the posterior columns. When the patient's foot was touched with the head of a pin, or when the point was pushed in slowly, it happened repeatedly (though the power of localisation was otherwise good) that the patient indicated with the greatest assurance the wrong foot—occasionally also indicating another as the corresponding point. This error was not committed however if the point of the pin was pressed in quickly and forcibly. As my attention was not directed to this point formerly, I am unable to say whether the same existed in the first two cases. But Fischer, to whom I shall refer below, has made some observations on this point.

Case IV.—N. Marie, ætat. 12½, of a neurosal family, some of the members of which have been epileptic. At birth the skull suffered compression by means of the forceps. She had typhus at the age of 3. The girl, who formerly had been very intelligent, became six months ago forgetful, absent, and complained of headache, at first in her forehead, and later in the occiput. She also had abdominal pain without derangement of digestion. These symptoms were regarded by her parents as the prodromata of the menses. At the beginning of February 1880, every evening attacks occurred in which, according to the parents' account, she fell into a state of stupor for one to two hours, during which she spoke unintelligibly, and did not answer questions put to her. Later on she had similar attacks also in the morning. During the intervals she had no recollection of the attacks. I saw the girl first on February 11. I found her in bed during one of her morning attacks. She was almost entirely unaware of her surroundings, playing with various toys, and only with difficulty made to put out her tongue, &c. The face was stupid-looking, and pale. The pupils were square, reacting slowly. There was no motor digest. Pulse 100. She specially delighted in drawing, but drew everything unnaturally and absurd. The attacks then assumed another character. The patient was restless, sang loudly, sprang on the table, &c., and then had muscular

spasms, which occasionally became opisthotonic. During the intervals the following condition was noted. In walking the right foot was dragged. There were spasms in the muscles of the neck and back, and especially when they were talked of; mentally dull, and though she could answer correctly, answered wrongly if admonished. Speech was somewhat like that of a child, but she could articulate well occasionally. She read unintelligibly and counted wrongly. She appeared not to recognise colours, but on strict cross-examination it was clear there was no colour-blindness properly speaking. The ocular muscles did not function altogether harmoniously. The two lower cervical and three upper dorsal vertebræ were tender on percussion. When asked to touch her nose she touched her ear, &c.

As to the cutaneous sensibility, it seemed somewhat diminished over the whole body. Asked to indicate the point where she was pricked in the right leg, she indicated it in the left. After this was repeatedly tried, I directed her attention away to something else, and during this pinched her with increasing severity in her left arm. Only as the pinching became severe she suddenly called out and laid hold of the corresponding part of her right arm.

At the beginning of April, under treatment, the patient had recovered, with the exception of occasional headache and restlessness.

An explanation of this peculiar symptom, allochiria, must be sought for in careful post-mortem examination, and cases like Case II. are desirable in this respect. At present I have not been able to obtain the requisite opportunities.

In the case mentioned we found extensive disorganisation of the lowest portion of the spinal cord. This, however, being transverse myelitis, was not well calculated to indicate clearly the paths of sensory conduction in individual portions of the cord. The complete sclerosis of the whole of the posterior columns for a considerable length above the principal lesion, gives a new proof of the correctness of the views of Woroschiloff, N. Weiss, &c., that the centripetal conduction of the spinal cord can take place without the posterior columns. It

is not however determined by these experiments whether the power of localisation is affected by destruction of the posterior columns or columns of Goll. It must be determined by future research in how far the occurrence of allochiria is related to extensive sclerosis of the posterior columns. As regards the ultimate signification of the small patches of degeneration which crossed the posterior horns I express no definite opinion, as we know so little of the physiology of this region.

The question occurs whether allochiria is an altogether exceptional occurrence in physiological or pathological states. Experience shows that the sensory and motor relations of the two sides of the body are not so independent of each other as is sometimes supposed. We meet rather with phenomena which indicate a close relation between symmetrical regions on both sides. As regards the motor association may be mentioned the well-known phenomenon of associated movements, particularly in paralysed limbs. By forced innervation both sides may be thrown into contraction without the assumption of simultaneous activity of both hemispheres. This bilateral action, so general in the new born, and also visible in certain muscular groups in adults, indicates a preponderance of the lower over the higher centres. Before specially referring to the sensory phenomena, I may mention the similar intimate relationship of the vegetative functions in both sides. As an example of bilateral vegetative function may be taken the activity of the great glands, which has been carefully investigated by Adamkiewicz.[1] Adamkiewicz and others (Adler, Asch, Buccola, Seppili, &c.) have also shown a similar bilateral relationship between corresponding tactile regions of the skin exemplified by the researches on transference of sensibility. The corresponding regions are for the most part antagonistic. If one side is hyperæsthetic, the sensibility of the other side is diminished. The point of importance in the present relation is the bilateral relationship here indicated. This is manifested especially in hysteria, and probably allochiria is occasionally to be found in this condition, as in Case IV. In the other three cases it was associated with organic spinal disease. As

[1] 'Die Secretion des Schweisses, eine bilaterale symmetrische Nervenfunction.' Berlin, 1878.

hysterical hemianæsthesia is similar in its symptoms to central hemianæsthesia of organic origin, so is hysterical like spinal allochiria.

Lately phenomena have been described which indicate not an antagonistic but a parallel relationship between corresponding sensory regions. Dumontpallier[1] has found that ether spray, applied to the left arm, caused also such anæsthesia of the right arm as to allow an abscess to be opened without pain.

I am unable to indicate how often allochiria has been met with in the numerous cases of disease of the spinal cord or of hysteria on record, but the following may be quoted as illustrations.

G. Fischer[2] has recorded two cases of tabes. In the one, in which the symptoms had appeared four years previously after a chill, it is stated that "the localisation of tactile impressions was very inexact. Strong pressure with blunt instruments were constantly referred to the foot not touched. This was repeatedly verified. The sensation of the prick on the great toe was retarded about four seconds. The reflex motion however occurred at the moment of the prick, or was occasionally somewhat retarded, but always occurred long before the sensation of pain," &c. Fischer also refers to another case in which the symptoms had come on a year previously. "Patella reflex very feeble on both sides, but stronger in the left than right. There were regions of tactile anæsthesia. The sensation of tickling was retained, and this gave rise to active reflex movements. Smooth objects held against the sole of the foot were felt as cutting instruments. The patient said also that when the right foot was touched, he also constantly felt something in the corresponding part of the left."

Hertzberg[3] describes a tabetic patient with very marked diminution in the power of localising impressions. "A touch of the finger on the feet could not be distinguished from

[1] 'Soc. Biol.,' Dec. 4, 1880.

[2] 'Zur Symptomatologie des Tabes Dorsalis,' 'Deutsch. Archiv f. klin. Med.' Bd. xxvi.

[3] 'Beiträge zur Kenntniss der Sensibilitäts-Störungen bei Tabes Dorsalis. Jena, 1875.

contact of a hard substance, such as a glass rod. Occasionally the patient said he had an indefinite sensation, but could not say whether the right or left leg had been touched, or whether it was the leg or any other part of the body."

Leyden[1] has also observed similar facts. In Case 28 the patient stated he could not perceive light contact with the finger. He felt needle pricks like a touch with blunt instruments. He had very uncertain power of localisation. He even referred a sharp prick with a needle on the left leg or foot, to the corresponding regions of the other limbs.

In another case (Case 27) "the localisation was very uncertain, so that the patient referred an impression on the one thigh to the other." In Case 7 "the sensibility was reduced to a minimum, the strongest pressure was scarcely felt; right and left were frequently confounded."

Leyden, in his 'Klinik der Rückenmarkskrankheiten,' remarks: "The faculty of sensory localisation is frequently so affected in tabes that foot, leg, thigh are confounded, and even when the legs are crossed mistake is made as to the limit touched."

The authors referred to are almost all disposed to attribute this sensory disorder, wholly or almost wholly, to the profoundness of affection of the localising faculty. But the first three cases I have recorded (the fourth may be omitted), as well as Leyden's Case 28, show that allochiria may be manifested when the power of localisation has only suffered to a slight degree, so that, with the exception of the error as to the side, the place touched was quite correctly indicated.

It would therefore be erroneous to look upon allochiria merely as a symptom of great sensory impairment. I am of opinion it must be regarded as a completely distinct symptom by itself. In favour of this also may be adduced the fact that in very many cases of central neurosis allochiria is altogether absent, notwithstanding the presence of a marked degree of anæsthesia.

I do not pretend to be in a position to offer a sufficient anatomical and physiological explanation of this peculiar symptom. But the following considerations may be advanced.

[1] 'Die graue Degeneration der hinteren Rückenmarks-Stränge.' Berlin, 1863.

Apart from the hysterical case, the allochiria occurred all in cases of disease of the spinal cord, mostly in tabes dorsalis. In my second case, which was of a different nature (compression-myelitis), there was, however, a marked degeneration of the posterior columns as shown by the autopsy. It is probable, therefore, as I have already indicated, that the degeneration of the posterior columns is the cause of the allochiria, though we are not yet able to indicate the more minute changes which lead to it.

The above clinical facts also bring out certain physiological points of interest. They show that there is an intimate connection, in a sensory relationship, between corresponding regions of the body, and also that an exactly isolated conduction in the sensory paths of the cord does not exist under all conditions, but under certain circumstances the sensory impulse leaves the accustomed paths and crosses to those of the other side.

THE HEART SYMPTOMS OF CHOREA.

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It is now generally admitted that there are certain heart symptoms proper to chorea apart from those which are due to its association with acute rheumatism or with anæmia. It is admitted as well that cardiac apex murmur is, more often than was at first believed, the sign of a functional disorder on the part of the valvular apparatus; the precise nature of this defective action has been in great measure elucidated, and it has been repeatedly shown both in chorea and other conditions that such murmurs are temporary and innocuous. With this double advance of knowledge, both in regard to the natural history of the disease in question, and the pathology of the heart, we may consider the cardiac symptoms of chorea from a new standpoint, with a clearer and more just apprehension than heretofore of the facts involved, and fuller and more definite material for their discussion. In what is to follow I propose to investigate the present state of opinion as to the nature and significance of the cardiac phenomena of chorea; to place such opinion side by side with the actual facts of the case; and upon this review, to consider whether by means of any existing hypothesis, or of any modification or combination of hypotheses, the question at issue finds reasonable solution.

The heart symptom of chorea chiefly discussed amongst us is systolic murmur. In regard to the rate and rhythm of the heart, as well as the precise seat of the bruit, there are, as we shall see, doubts and differences; but all observers allow that a soft systolic murmur is apt to arise in the course of chorea, independently of rheumatism or of anæmia, which is variable in tone, disappearing and reappearing, often influenced by

posture and by exercise, most audible in the majority of instances at the left apex, productive of no sensation of its own, and indicated by no outward sign of disturbed circulation.[1] Between this cardiac murmur and the choreic restlessness there is no equality or parallelism. No particular manner or degree of mismovement, and no particular temperament of patient is especially liable to this heart affection. The choreic murmur comes and goes secretly and without notice, there is no recognisable law of its occurrence, and when auscultation has discovered it, neither treatment nor prognosis is greatly influenced by the discovery.

But while these symptoms on the part of the heart, in their physical characters, limited duration and freedom from injurious consequences, remind us most of the so-called functional murmurs, yet have they a near relationship to substantive disease. This is not matter of inference, but of direct observation. There is no fact of chorea better established than this: that in the majority of fatal cases—dying either in the course of chorea or shortly afterwards—a fibrinous bead-like fringe is found edging the mitral valve, and occasionally the aortic valve also.[2] That this condition is the effect rather than the cause of the chorea, we are compelled to assume, in the numerous instances where murmur has gradually developed in the course of the muscular disorder. There are others, however, and especially those which arise out of acute rheumatism (be they few or many), where the sequence is different, and the cardiac murmur, from immediately preceding the chorea, may be taken to indicate an endocarditis which is certainly not the consequence and may possibly be the direct cause of the chorea.

But although endocarditis, upon this showing, seems to be connected with chorea in a double relationship, both as its cause and its product, there are obvious difficulties in the way of admitting it at all. For if endocarditis is really at the origin of chorea, why do the physical signs which should

[1] Such is the general description. Dr. Walshe, however, insists upon the regularity of action, and Dr. George Balfour asserts that the murmur "while it lasts, is constant and unchanging." See Walshe, 'Diseases of Heart,' p. 89; Balfour, 'Diseases of Heart,' p. 166.

[2] 'Statistics of Fatal Chorea.' 'Lancet,' July 17, 1880. 'Pathology of Chorea.' Dickinson, 'Med. Chir. Trans.,' vol. lix.

announce it so often lag behind, and why are they in themselves so equivocal and without the usual accompaniments and results of valve-inflammation? If, on the contrary, endocarditis is not the origin of chorea, but arises out of it, by what sort of agency is this accomplished, and why does not chorea, in virtue of this association, lay the foundation of organic heart disease as acute rheumatism does? Two conflicting doctrines are thus offered for our acceptance, neither of which we can wholly accept, and neither wholly reject. It is certain on the one hand that cases of chorea which happen to die (whether from the severity of the disorder or otherwise) very commonly exhibit a fringing of recent lymph on the mitral valve; it is not less certain on the other that the non-fatal cases (that is of course the vast majority) very rarely exhibit valvular disease in later life. It is certain that both the auscultation signs, and the subsequent history of choreic murmur correspond in most respects with the dynamic murmur of young women; it is not less certain that chorea prefers children rather than young women, and shows after death the actual material of endocarditis.

Such are the difficulties which meet us at the outset in endeavouring to explain the cardiac symptoms of chorea, and it is obvious that the account I have just given in regard to these symptoms does not contain the key to the solution. There still remain, however, other factors of the problem, some which are differently interpreted by different observers, and some which perhaps have not as yet received their due share of notice. Thus, for example, there is the character of the heart's rhythm, which some report to be irregular, and others undisturbed; and there is the further question as to the mode of incidence of cardiac phenomena in chorea in respect of age. It may be that the discrepancies which appear at present in regard to the precise characters and relative frequency of heart disturbance will disappear when we come to compare patients of similar age. We know, for instance, that the liability to such disturbance is in no direct relation to the violence or the particular method of the choreic attack; but we do not know that it has no direct relation to the age of the patient. Again, we know that choreic murmur may occur in-

dependently of any marked change in the cardiac rhythm; but we do not know that this is so at all periods of life, or that disturbed rhythm is not the rule rather than the exception in young children.

In what follows I propose to consider the characters and the frequency of cardiac disturbance in young children as contrasted with those who are older; to compare actual observations upon these points with the more general statements of authors; and, by the help of this comparison and the material it will require, to consider what hypothesis serves best to reconcile phenomena which at first sight seem to be conflicting. I take for this purpose the following:—

1. Dr. Dickinson's seventy-one cases (not 70 as stated) appended to his paper in the fifty-ninth volume of the "Medico-Chirurgical Transactions," and referring to children treated at the Great Ormond Street Hospital.

2. One hundred and thirty-two cases of children treated by myself at the same hospital, but at a late stage of their disorder.

3. Thirty-nine cases of older children and young adults under my charge at the Westminster Hospital.

4. Fifty cases similar to the last recorded by Dr. Owen in the ninth volume of the 'St. George's Hospital Reports.'

These four groups represent respectively, as will be seen more particularly in the sequel—

(1.) Young children from 2 to 12 observed at an early period of their disorder.

(2.) Young children of similar age observed at a later period of their disorder.

(3.) Older children with a few adults.

(4.) Older children, with a considerable proportion of young adults.

In Dr. Dickinson's table the number has to be reduced to 69, on account of two cases with the ages omitted. The children are all young. There is in fact but one, a girl, as old as 13, and but 3 as old as 12; the youngest is 3, the next youngest are 2 of 5; the ages most largely represented are 10 and 11. Taking the 69 cases, with a view to ascertain the incidence of heart affection at different ages, it will be found

that of the 21 children who are 8 years old and under, only 4 are free from heart affection, 3 of these being of the age of 8;[1] while of the 48 children above 8 years old, there are 17 free from heart disorder. If, however, we state the numbers somewhat differently, classing the children of 8 amongst the elder ones, we shall then find that in 11 children under 8, there is but one free from heart affection, while in 58 of 8 years old and over, there are at least 19 free from any abnormality of cardiac action which can fairly be reckoned.[2] As regards irregularity, the 69 cases give 20 such examples, 12 of irregularity alone and 8 of irregularity together with murmur. Of the 21 children 8 years old and under, there are 5 with hearts irregular only, and 3 with irregularity together with murmur. Of the 48 children over 8 years old, there are 7 with hearts irregular only, and 5 with irregularity together with murmur.

I take next a second group of children from the same hospital who were under my own charge, having been received from one or other of my colleagues.[3] The ages are still between 2 and 12, as in the first group, but the children come under observation at a later stage of their disorder; the great majority were chronic cases, some few were convalescent. The total number of these patients was 132 (appearing as 137, from 5 of them having been twice admitted). The age distribution is as follows:—

12 years old are only	2
11 years old are	24
9 and 10 years old	47
8 years old and under, 36 of these being under 8 . . .	59
	132

[1] Of the 17 children of 8 and under having heart disturbance as many as 5 had had acute rheumatism, to which therefore the heart condition might be referred. On the other hand, of the 31 children over 8 having heart disturbance, as many as 6 had had previous attacks of chorea, to which period therefore rather than to the age recorded, the heart disturbance might be referred.

[2] I have made these calculations from the tables themselves (to which I refer the reader), and they do not precisely correspond with Dr. Dickinson's own summary (on p. 34 of his paper). This latter, however, has reference not to the *ages* of the patients, but to the several *causes* of the chorea.

[3] I have referred to this series elsewhere, in connection with the rheumatic origin of chorea. 'Lancet,' Nov. 29, 1879. It is not given here *in extenso* out of regard to space.

In the whole 132, 47 (more than a third) have heart murmur or irregularity. The 59 children of 8 and under have 24 examples of heart disorder; the 72 children over 8 have 23 such examples. In other words, the proportion of heart disturbance is about 40 per cent. in the younger children, and about 32 per cent. in the older ones.

Again, if we take the extremes of age in both directions, that is to say, if we compare the 26 children of 11 and 12 (the oldest we have to do with) with 36 who are *under* 8, we have 9 of the former having heart disorder against 16 of the latter, the actual percentage of heart disturbance being thus somewhat greater in the younger than in the older children. Of cardiac irregularity, as apart from murmur, not including mere unevenness of rhythm, there are but 9 examples, 6 of these referring to the younger children.

These cases, therefore, representing the same ages as Dr. Dickinson's, but referring to a later stage of the affection, yield a much diminished proportion of heart disturbance of whatever kind. It is a little over a third, whereas in the former table it is over two-thirds. And what is still more remarkable, irregular action of the heart, largely represented in Dr. Dickinson's list, hardly appears in mine, and is almost confined to the younger patients.

It thus appears probable, taking these two tables as they stand, and comparing them together, first, that the heart affection of chorea is not less but more marked in early than in later childhood; secondly, that observation of the disorder at a late period discovers less heart disturbance than at an earlier, and especially that cardiac irregularity, a distinguishing feature of the chorea of early childhood, is found at the beginning rather than at the end of the attack.

Such, I say, are the conclusions to be drawn from a bare enumeration, and so long as we confine ourselves to children the majority of whom are not over 10, they are conclusions which need no correction. But when we enlarge the view and admit older children and adults; in other words, when we go from children's hospitals to general hospitals, we can no longer accept the mere figures without some attempt at adjustment on account of disparity of age. Thus, for example, all other

things being equal, the greater the age the larger the proportion of heart disturbance independent altogether of chorea. We have to consider besides, that in comparing childhood with adolescence, in respect of the signs we are considering, we have to encounter with the later period those functional murmurs (variously named, and attaching especially to young women) which, although they be altogether distinct from chorea as such, are not in practice distinguishable from it. Add to this that the older the patient the greater the probability that the chorea is not the first, but the second or the third attack, and the heart disturbance therefore but a revival of what has occurred in childhood, according to the well-known rule of the disorder to repeat itself. Such cases illustrate in fact the liability of *early* childhood in respect of heart sympathy: they are taken to illustrate the liability of the more advanced age at which the patient comes under notice. Again, some account must be taken of the previous occurrence of acute rheumatism, which is likely to appear in larger proportion in the histories of the older patients than of the younger, and to contribute its share of heart murmurs which are not choreic but rheumatic. It would be quite unfair, I say, to attempt the comparison we are now about without making allowance for discrepancies of this kind; without providing, that is to say, for the fact that the younger the child the more likely the heart disturbance to be intrinsic, the older the child the more likely to be accidental or contributed. In any exact computation as to the age-incidence of heart disturbance in chorea, it would be necessary of course to exclude from the comparison anæmic and chlorotic girls, as well as those who in earlier childhood had had either chorea or acute rheumatism. In the case of such children as we have been considering, all of them young, the correction necessary on this account would not amount to much, and we need not stay to make it; but in the case of the older patients we are now coming to, it makes a very wide difference. It would be altogether delusive and misleading to place the old and the young side by side with the view to an even comparison in respect of heart sympathy in chorea. Numerical equality between the two classes, or anything near an equality, would

indicate, without doubt, that the heart was more often implicated in the chorea of the young than of the less young.

How the figures stand in these respects with older patients, and how large a correction is needed for such reasons as have been mentioned, will be seen from the examples of chorea which remain to be quoted, viz., the 39 of Westminster Hospital and the 50 of St. George's Hospital. In the 39 Westminster cases 20 are 12 years old or over, 5 of them being young women. In the 50 cases reported by Dr. Owen in the 'St. George's Hospital Reports,' vol. ix., of two following years, as many as 27 are 12 years old and over, 11 of these being young women and 6 young men. We have thus in the four series of cases now rehearsed a constant rise in the ages of the patients. Dr. Dickinson's are the youngest, and the St. George's cases are by much the oldest, the Westminster series standing midway, and my own Children's Hospital cases differing from Dr. Dickinson's more in the duration of attack than in age of subject.

The 39 Westminster cases are distributed as follows:—

13 years and over (2 being young women)	15
12 years old	5
11 years old	5
9 and 10 years old	9
8 years old and under	5
	39

Of the whole number, 13 would have to be excluded, 5 on account of acute rheumatism at some earlier period, and 8 on account of previous attacks of chorea. The remaining 26 are made up of 19 who have no cardiac disorder, and 7 who have either murmur or irregularity. Of the 19 who have their hearts free, 15 are over 11 years old; of the 7 who have cardiac murmur or irregularity, only 1 is over 11.

In the two tables of cases of St. George's Hospital there are 15 examples of heart disturbance in 50 cases, 10 of these 15 are found among the 27 cases over 12 years old; only 5 are found among the 23 cases under that age. Upon this showing, it would seem that cardiac symptoms in chorea are not more rare but much more common after 12 years old than before.

Upon examination, however, of the 10 individuals exhibiting heart murmur and over 12 years old, it appears that 4 of them have had previous attacks of chorea, 1 had pericarditis while in hospital, 1 developed murmur just after acute rheumatism, and 2 were weakly girls, aged 14 and 15 respectively. There are thus 8 of the 10 whose murmurs are accounted for apart from chorea altogether. We get but two properly choreic murmurs in 27 choreic patients of ages ranging from 13 to 18, as large a proportion, I am disposed to think, as will be commonly found at that period.

There is indeed considerable difficulty in fixing the time of life when the liability to heart disturbance in chorea finally ceases. The evidence already adduced shows, I think, that childhood is particularly liable to it, and that irregularity of rhythm distinguishes this earliest liability, whether with or without systolic murmur. It is also generally agreed that the rare chorea of advanced life has little connection with heart disturbance. But of the middle period our knowledge is far from precise. For it so happens that at the very time when, with the progress of development, the frequency of cardiac murmur in chorea is obviously lessening there comes a period of life (and especially with young women) when the precise significance of the symptoms we are discussing is hardly discoverable. Murmur may be choreic or (so-called) anæmic. It would seem impossible at such time to determine with any approach to certainty the proper liability of the heart in connection with chorea. We know only that so soon as this interval of obscurity has passed, and, with the completion of adult life, the indications furnished by the heart are again trustworthy, cardiac sympathy in chorea is hardly recognisable.

From the facts now adduced several conclusions seem to follow. Heart symptoms in chorea occur most frequently during a period which is included between 2 years of age and about 7 or 8. The age of greatest liability to choreic heart disturbance thus partially coincides with the age of greatest liability to chorea. Again, the abrupt decline in the liability to chorea, which occurs at about 15, corresponds with a similar decline—if it be not rather a cessation—of the dispo-

sition of the heart to share in the disorder of the voluntary muscles.

It has been observed already that irregular cardiac action, either alone or preceding or accompanying the murmur, is a pretty frequent feature in the younger children of Dr. Dickinson's table. In my own children of similar age, but in a later stage of chorea, it is much less common. In the Westminster cases, where the patients are older, it is also rare, and in the St. George's cases, older still, irregularity hardly occurs. This particular symptom, in the younger subjects, is often the single sign of the heart's affection. I believe, although I cannot show by tables, that there are very few little children, if there be any, in an early stage of chorea, who do not exhibit in some degree unevenness of cardiac rhythm. Such unevenness will sometimes develope into murmur and sometimes not, murmur which is almost always variable with position, sometimes best heard over the pulmonary cartilage rather than at the apex,[1] apt to come and go, and often finally lost sight of before the chorea itself has disappeared. The second pulmonary sound is seldom (Dr. Walshe says never) either accentuated or doubled.

These statistics in regard to the age-incidence of the heart symptoms of chorea enable us to render a fuller account than that first given of the particular phenomena. Dr. Walshe's assertion that the cardiac action is regular must be limited to the older subjects, or to the later stages of the disorder. Dr. Bristowe's statement that the greater number of choreic patients have some cardiac defect, irregularity amongst the rest, must be qualified by adding that this large proportion refers to children, and that irregularity as a sign of heart disturbance is almost confined to them.

[1] The fact of mitral regurgitation being most audible in this situation, is well explained by Naunyn, as reported by Rosenstein (Ziemssen's 'Cyclopæd.' vol. vi. p. 122.) He points out that the second intercostal space, close to the left edge of the sternum, coincides with the point of the left auricular appendix, which winds round the pulmonary artery and lies in front of it. Now with the abnormal current of blood flowing towards the auricle, we can easily understand, says Rosenstein, "how the sound should be conducted to the spot mentioned better than towards the apex, more especially in cases where the appendix is long enough to lay its point close against the anterior wall of the thorax."

Thus the several factors of the problem with which we have to deal summarily stated are these:—

1. In the course of the chorea of childhood the heart is apt to become irregular or uneven, and its first sound to be followed by apex murmur, which is variable in pitch, influenced by posture, seldom audible in the axilla or at the angle of the scapula, and which disappears along with or shortly after the chorea, the heart and the circulation suffering no injury.

2. This liability on the part of the heart to what, from its signs, would seem to be a functional disturbance is independent of the violence or method of the chorea, but dependent upon the age of the patient, the younger children being the most, and the elder the least liable, while beyond childhood there is little if any liability of the kind.

3. These heart signs of chorea—acute rheumatism being excluded—give rise, as a general rule, to no symptoms whatever affecting the health or comfort of the child. They make no apparent difference to the prospects of recovery or the structural integrity of the heart. Nevertheless, choreic children having this murmur and happening to die, either with or shortly after recovery from chorea, very commonly exhibit a beading of recent lymph on the mitral valve.

Such, I say, are the chief statements which statistics seem to warrant. I will venture to add another, which, so far as I know, has never been statistically reckoned, but which no one will gainsay. It is, indeed, the most constant of all the heart symptoms of chorea, and met with at a later age than the rest. I mean the acceleration of the heart and pulse.[1]

Now there are two hypotheses of the heart phenomena of

[1] Ziemssen says "cases of arhythmia are certainly very rare, and there is hardly anything published about it." On the other hand, he regards acceleration of the action of the heart as common, and acceleration of the pulse as quite constant, although, as he believes (I am convinced erroneously) the bodily heat is not changed even in severe cases. (Ziemssen's 'Cyclopæd.' vol. xiv. art. Chorea, p. 436.)

The heart symptoms mentioned in the text are not the only ones to be met with in chorea. I have heard presystolic murmur, and both accentuation and doubling of the second sound, and this in cases where there was sufficient evidence that the heart disturbance was purely choreic. But I am not able to say at present to what ages these exceptional signs chiefly attach, or what is their proportion to the commoner symptoms.

chorea, each of which has special regard to certain of the facts just mentioned, although neither is able to reckon with them all. The two are, in a sense, mutually antagonistic, inasmuch as what the one rejects the other prefers. Both theories have their supporters in this country. The one asserts that the heart symptoms of chorea are due to endocarditis, the precise relationship of the muscular disorder to the valve inflammation being variously interpreted. The other asserts that the cardiac signs are intrinsic, a part of the chorea itself. That there is some evidence in favour of each of these views we have already seen. The task before us is to reconcile them.

To the belief that the heart murmur of chorea is due to endocarditis I know of nothing in the actual clinical symptoms that can be opposed. Our information in regard to the physical signs of endocarditis *per se* is, indeed,[1] vague enough, while the probability of its occurrence in chorea is directly favoured, as we have seen, by post-mortem evidence. The distinction between organic and functional heart murmurs, founded on hard and fast lines as to the transmission of the bruit, is of doubtful validity. "A well-pronounced functional murmur," says an excellent observer,[2] "may be as diffused and transmitted as an organic." "A systolic apex murmur," says Dr. Bristowe, "is a positive proof of regurgitation." We have indeed, just as much evidence, so far as physical signs are concerned, of endocarditis in the course of chorea as we have of it in many instances of acute rheumatism. In both cases alike a soft and not conveyed murmur may be proved, in the event of death, to depend upon valve deposit, and in both

[1] "There are few diseases," says Rosenstein, "the presence of which is diagnosticated so arbitrarily as that of original acute and subacute endocarditis." He proceeds to point out the necessity of "proving the existence of *other symptoms in addition to the mere murmur*, especially intensified second sound in the pulmonary artery, the localization of the murmur, or transverse hypertrophy of the cardiac volume." Rosenstein, art. Endocarditis, Ziemssen's 'Cyclopæd.,' vol. vi. p. 90. Dr. Bristowe says, 'If in the progress of any of those diseases of which endocarditis is a common complication, we detect a recent cardiac murmur, and *if further observation proves this to be a permanent phenomenon*, we cannot reasonably doubt that endocarditis is present." (Bristowe, 'Theory and Practice of Medicine,' 2nd ed. p. 522.)

[2] Dr. Nixon in 'Dublin Journal of Med. Science,' vol. iv. p. 575.

alike (I do not say to a like extent), when death does not so intervene, the murmur has been found to disappear. In frank recognition of these facts, Dr. Wilks expresses the belief that all mitral systolic murmurs associated with chorea are organic; and Dr. Sansom,[1] in a short summary of the prevailing opinions upon the subject, admirable for its conciseness, arrives at a like conclusion.

But when this much has been granted and endocarditis is admitted, how much are we the better? What is the mode of origin and what is the sequel of such endocarditis? It is but exchanging one difficulty for another. For we have to suppose, upon this hypothesis, not only that endocarditis may arise in and out of chorea, and that the younger the child the greater the probability of this event, but also that cardiac irregularity sometimes precedes and sometimes takes the place of regurgitation, whilst none of the injurious after consequences which attend endocarditis in its other relations are found to ensue here. In face of such grave objections, we are forced to admit that, although physical signs do not contradict it and post-mortem evidence may seem to be directly in its favour, the theory of choreic murmur (not to speak of the other signs) which invokes endocarditis is too difficult. It is inconsistent with all that we know of that inflammation in its various pathological relations; and apart from these we know remarkably little about it.

It is in fact by virtue of this vague knowledge that endocarditis maintains its hold in this connection. A form of heart inflammation special to chorea, and having a history and mode of termination of its own, is a creation of too shadowy a kind to be easily dealt with. But so soon as anything definite and within reach of investigation is alleged in regard to it, when, for instance, it is said (as Trousseau, Bouillaud, Roger, and other French authors say) that the endocarditis of chorea is in fact rheumatic, although the rheumatic element is itself latent, we have a distinct yet still but partially tangible issue. Of the relationship between chorea and rheumatism I do not propose to speak in this place. I have endeavoured to show elsewhere that although real it is rare and exceptional, and

[1] Sansom, 'Diseases of the Heart,' p. 89.

both Walshe and Hayden are of the same opinion. However this may be, no one will refuse to admit that the cardiac symptoms of chorea are observable sometimes when there is no open rheumatism either past or present. Now if in all such instances we are to say, in the first place, rheumatism is actually present, but after a secret and ineffable manner, and in the second place endocarditis is the consequence of that rheumatism, but it is an endocarditis which is exceptional in commencing after the chorea has begun and disappearing along with it, we account for the facts no doubt, but after a fashion which rather begs the question than provides for it.

We are thus compelled to regard the heart symptoms of chorea, taking them in their entirety and not by arbitrary selection—the early irregularity, the variable mitral murmur, the very frequent acceleration of heart and pulse with frequent fluctuations in the rate of both—as being altogether special and peculiar. They are signs of the heart's sympathy with the voluntary muscles, and are seen most at that early time of life when the antecedence of acute rheumatism is the least probable. The heart suffers by whatever mechanism or nervous influence, part and parcel with the rest of the muscular system, or rather it is apt to suffer. Such a conclusion, I say, in some sense or other, seems self-evident, and is indeed involved in the statement that the heart symptoms of chorea are met with in no other association.

Obvious and inevitable as it may be, it is far from being easy of application. For if it be granted, in the words of Dr. Walshe, "that the apex murmur of chorea is plausibly ascribable to disordered action of the muscular apparatus connected with the valve," why does not the same disordered action extend to the ventricular walls? It may be answered (although Dr. Walshe does not admit as much) that in young children there is both disorder of rhythm and regurgitation; but the answer is insufficient. These two signs do not commonly concur nor do they vary together. And it is not young children alone that we have to consider, but the elder patients as well; and in these latter, while murmur is not infrequent, any marked irregularity of cardiac rhythm is

certainly rare. Thus we have to suppose that the fibres of one and the same muscle are acting regularly as regards the ventricular wall, but irregularly as regards the papillary muscles. "We should be forced to admit," says Dr. Hayden, "that whilst the greater portion of the length of certain muscular fibres contracts with perfect order and regularity, the remaining and smaller portion of the same fibres acts spasmodically and out of harmony with the former. No physiologist," he adds, "bearing in mind the unity of nerve centres and the community of nerve distribution enjoyed by both portions of the same fibres, would admit such a doctrine."[1]

There are other objections equally formidable. It is not in the nature of chorea to dissever the normal combinations of muscular movement. It stirs the very same muscles that are employed by purpose or emotion, and moves them in a similar way, only less precisely and with less definition. Apart from anatomical considerations, therefore, a choreic spasm of the heart, which should select the papillary muscles and leave the cardiac walls, would be untrue to the pattern of chorea elsewhere. And again, if this choreic conduct of the heart be but an extension of the same disorder which affects the other muscles (an extension, as Dr. Kirkes points out, to which there is no parallel in any other involuntary muscular organ), the most violent and general chorea ought to exhibit this cardiac disturbance the most. But it is not so. On the contrary, the violent chorea of puberty has seldom either cardiac murmur or cardiac irregularity, while the chorea of the young child, which is but rarely violent and is sometimes rather a paresis than a disorder of movement, exhibits both these forms of heart affection very often.

And even if these difficulties should be overcome or the ground of some of them disputed, it would still, I think, be hard to understand how the great centre of circulation and (in one sense, at all events, within the experience of everybody) of emotion should become the seat of a mismovement at all resembling the choreic contortions of the visible muscles without giving rise to sensations of grave disquiet, sensations, be it remembered, which, so far as young children are con-

[1] 'Diseases of Heart and Aorta,' p. 268.

cerned, the favourite subjects of such cardiac disorder, are conspicuously absent.

It would thus appear that both the hypothesis which supposes endocarditis to be the cause of choreic murmur and the hypothesis which supposes an extension of the muscular infirmity to the heart to be the cause of it are fallacious. Both theories commit us to too much. And if, in this strait, we have recourse to vaguer expressions having regard to the "impoverishment" or "vitiation" of the blood, we neither provide an adequate explanation nor allege a demonstrable cause. But while thus compelled to abandon one position after another, we must beware lest by too precipitate a retreat we quit ground which it is essential to hold. We may even venture so far upon the assured facts of the case as to make some sort of stand and to affirm something. If the views just announced cannot be held in their entirety they must be held with some modification or other. There is no alternative. Part of the perplexity arises, as has been shown, from a consideration of the harmlessness, variability and limited duration of the choreic murmur. But if these facts are in themselves secure we may rely upon them to force at least this conclusion, that the conduct of the heart in chorea is similar to its conduct in anæmia. Whatever explanation, therefore, applies best to the precise mechanism of the so-called functional or dynamic murmur cannot be altogether foreign to the circumstances of chorea.

It is now generally admitted that the murmurs once called hæmic, anæmic, and by similar names implying dependence on some blood change, are much more often than was at first supposed produced by actual regurgitation, due to a temporary defective action on the part of the mitral valve. Such murmurs are to be heard in purpura, in anæmia, in the general muscular relaxation after various nervous strains, in epilepsy, as well as in typhus and enteric fever, and even in simple pyrexia. This dynamic mitral bruit may, by exception, be harsh and far sounding, audible in the axilla and even at the lower angle of the scapula, yet nevertheless by its ultimate disappearance give ample proof that whatever may befal the valve during its continuance (and there is some reason to believe that a material change actually occurs) is not abiding

or permanently mischievous. So much, I think, is generally admitted. The question for us now is, whether the same explanation of the mechanism of this murmur which applies to its association with the conditions just mentioned applies also to its association with chorea.

No one, so far as I know, has more ably discussed this subject than Dr. Nixon.[1] His exposition of the various ways in which dynamic apex murmur may arise, and of "the perfect unity and correspondence in action of the fibres of the walls of the ventricles and of those which are connected with the valves, which is needed in order to ensure complete closure," suggests to him the inquiry whether this nice correspondence of movement might not be so disturbed by altered nutrition or innervation as to render such closure for a while defective. The tremor of the voluntary muscles, which is apt to follow upon their violent exercise, is adduced as being analogous to this disordered cardiac action. Dr. Nixon concludes that functional mitral murmur results from "this want of correspondence between the fibres of the ventricle, which obliterate the cavity, and those which close the valve;" an altered function due, as he believes, "to some defect in the vital power or condition of the heart itself, which leads either to atony of the papillary muscles or derangement in the rhythm of their movement." In the further observations of the same author as to the means of separating this murmur from the organic, he alludes to its changeable character, alteration or even disappearance with the upright posture, and the absence (in most instances) of accentuated or double second sound. Thus, in all its essential features, the murmur which Dr. Nixon describes corresponds with that of chorea; and, except in cases of temporary debility, anæmia and certain conditions already particularised commonest in the female sex, it is seen in no other connection.

Dr. Hayden, assenting in part to these views, would attribute the dynamic murmur not so much to a want of correspondence between muscular fibres, as to atony or parietal debility of the walls of the left ventricle.[2] Weakness and relaxation of the papillary muscles, he admits as a possible cause of mitral regur-

[1] 'Dublin Journal of Medical Science,' vol. lv. p. 372, &c.

[2] Hayden, loc. cit. p. 274.

gitation. Spasm, or irregular action of the same muscles, he does not admit as a possible cause of it. This regurgitant murmur, "caused by atony and partial yielding of the walls of the left ventricle at the acme of systole," Dr. Hayden has long associated with anæmia, purpura, and excessive use of tobacco. His later observations lead him to include chorea and fatty heart in the same category.

But the latest and least reserved expression of the opinion that dynamic murmur is in fact dependent on muscular paresis is that of Immermann.[1] "The muscular tissue of the heart," says he, "owing to the altered state of the blood, is easily fatigued, and this liability to premature fatigue extends to the papillary muscles connected with the auriculo-ventricular valves. After any undue exertion of the cardiac muscle a temporary paresis of the musculi papillares ensues. In consequence of this the valve flaps intrude into the auricles with every ventricular contraction, that is, a transient functional insufficiency of the tricuspid and mitral valves is established. As the organ regains its normal energy these grow fainter and ultimately disappear." He goes on to describe the fatty degeneration of the papillary muscles which sometimes ensues upon simple anæmia, and which explains both the occasional permanence of these murmurs and their occurrence in connection with fatty heart.

It will be admitted, I think, that both the time of appearance and precise manner of the choreic murmur, as well as its mode of origin and duration, varying intensity and limitation to the period of youth, are all nicely provided for in the hypotheses just rehearsed. If only it be admitted that the heart's disorder is really a sympathy with the muscular disturbance, an inexact and precarious correspondence between the ventricular contraction and that of the papillary muscles would seem to serve for chorea even more than for the conditions to which Dr. Nixon applies it. Still better, and without making any assumption as to the heart's sympathy, might we admit the doctrine of partial paresis of the valve apparatus, and insist that the excessive muscular movement of chorea is the precise condition which, upon the hypothesis, should give rise to

[1] Immermann, art. "Anæmia," Ziemssen's 'Cyclopæd.' vol. xvi. p. 399.

murmur such as we hear in token of fatigue on the part of the heart. Adopting either of these views, we might say with Ziemssen that "change in the valvular sound is *à priori* more likely in chorea than in any other affection."[1]

But the matter is not so easily settled. Let it be granted that the choreic murmur is due to a functional disorder on the part of the mitral and probably also of the tricuspid valves, by means of which a temporary and variable regurgitation is permitted (and I think so much of admission is inevitable), we have to ask, in the next place, why this regurgitation, whether it be due to faulty adaptation of the several parts of the heart to one another, or to the paresis of fatigue, is not in harmony with the other symptoms? If the murmur betokens a sympathy on the part of the heart with the disordered action of the other muscles, it ought to appear when chorea is the most generalised; if, on the other hand, it betokens a fatigue paresis, it ought to appear when the chorea is most violent and prolonged. But it does neither. The sympathy of the heart (if it be rightly so called) goes not with the manner or the degree of the chorea, but, as had been shown, with the age of the patient. And, moreover, we should be taking a very partial view of the subject were we to limit the heart symptoms to cardiac murmur. This, as we have seen, is but one, and not even the chief, symptom of choreic heart disturbance. There is besides the unevenness or irregularity which belongs especially to early childhood, and, most constant of all, there is the accelerated pace of the heart. Again, we have not merely to account for the presence of these additional signs, we have to fit them into their places, and to show why the heart, seldom failing to share the muscular disorder in one way or another, exhibits this mode of disturbance or that according to the age of the subject. We may accept either account of the mechanism of dynamic murmur, and without much violence apply it to suit the circumstances of chorea: that is a little gain, no doubt. But when that is done there remain these further symptoms, which, unlike valvular murmur, can compare with nothing to be found in other analogous conditions, unless indeed the cardiac unevenness and its acceleration remind us of what is sometimes met with in

[1] Loc. cit. p. 440.

hypochondriacs and hysterical women, the least promising subjects to throw light upon obscure places in pathology.

It would seem then that to understand the conduct of the heart in chorea we must look to chorea itself and the modifications it exhibits at various ages. Here is an affection which both in absolute frequency and in its tendency to implicate the heart, attaches itself to young children most of all, to older children somewhat less, to adults hardly at all. At each of these periods chorea, both in the limbs and at the heart, shows itself after a particular manner. If, then, the action of the heart in chorea is throughout in real harmony with that of the voluntary muscles, the clue to the secret we are in search of must be sought in the special characteristics of those periods of life to which chorea especially attaches—characteristics, that is to say, in which childhood and youth differ from infancy on the one hand and adult life on the other. If the reader is not already wearied by the length of this exordium and the somewhat circuitous path we have had to pursue, I would venture in such space as remains to take a final survey of the subject from this point of view.

I would observe, in the first place, that the chorea of the post-infantile period is the immediate successor of the muscular spasm or convulsion of the infantile period. At an age varying with the "forwardness" of the child, the one liability takes the place of the other. The very same material cause (as for example, intestinal irritation, or the troubles of dentition) will excite convulsion in infancy, and chorea a little later. Again, the chorea of early childhood will sometimes commence with convulsion, or the two affections may alternate, the same limbs being affected in both cases. Little, of course, can be certainly known as to the actual sensations of the child at this early age, or of the mode in which physical pain gets mentally interpreted; but it depends probably in some measure upon the degree of completeness of such interpretation whether the response shall be by way of convulsion or by way of chorea. However this may be, the general muscular restlessness of the young child which passes for chorea is not only in close union with spasm in the way just mentioned, it often bears a direct resemblance to it in form. There is a uni-

versal unsteadiness in which the muscles of the abdomen and of respiration share as well as the diaphragm and larynx. This generalised chorea which thus succeeds to convulsions is, so to speak, the simplest form of the disorder, without variety so far as the movements of the several parts of the body are concerned, and without any mental admixture. It is an aggravation of the natural unsteadiness of childhood, an universal and equable overmovement or mismovement extending to all those regions which it is the nature of chorea as a motor disorder to affect, and it is this merely. There is no incoordination, for the muscles have not yet learnt to act in concert; there is no distinction between the movement of one part and another, for the several muscular uses are not yet differentiated, and the limbs are stirred indifferently without method or purpose. Such general, featureless agitation of the body is the proper response to a source of irritation which appeals to no one part in particular and has no mental representation.

With maturer age the bodily movements become varied and definite, and each department of the muscular system sets about its separate business. And as with movement so with mismovement. The chorea of later life is a definite deformity which alters and disfigures the natural carriage in this way or that according to its seat. It selects those muscles which are the readiest to respond to emotion, and moves them as they are wont to be moved by the passions and employments of life. Thus the choreic distortion of the face is one thing, of the legs another, of the arms another; and this separateness becomes the more distinct as the individual grows and his individuality, as it is called, becomes more and more marked. The mismovement of chorea betrays the age of its subject, no less than does the natural movement. And as life goes on chorea becomes more and more distinctly localised. In its latest form it is a partial disobedience or misconduct of certain muscles, rendered inveterate by habit, and incorporated as it were with the rest of the bodily movements.

My conjecture is that the conduct of the heart in chorea is in strict accordance with these successive modes or differentiations of the muscular disorder. The heart sympathises with

chorea in one way or another throughout life, or at any rate until that period is reached when the affection is both rare and difficult to identify, and has ceased to apply to the whole system. Not to speak for a moment of the valvular murmur, the heart is unrhythmical or irregular in early childhood,[1] accelerated in youth and left undisturbed in old age. Thus the earliest period of heart sympathy, that of irregularity, with or without acceleration, corresponds with chorea, a purely motor disorder, the earliest transition from infantile convulsion and differing from it mainly in the character of the movement and the implication of the emotional centres; but resembling it in its exciting cause, wide range, and conformity everywhere to a single pattern. In the heart as well as the limbs, in the tongue, the speech, the diaphragm, in all the parts in fact accessible to emotion, yet without any mental representation of emotion, the choreic disturbance of little children is a motor disorder, merely excited very often, like the earlier convulsion, by a material irritation.

In like manner the cardiac acceleration of youthful chorea, with or without murmur, corresponds with that form and period of the affection when the several muscular movements have been differentiated, and there is no longer a single and uniform response, but each department of the body liable to chorea responds after its own manner to a disorder which is now first mentally apprehended and intimately connected with emotional exaltation. As to the limbs there is no longer a mere restlessness impartially distributed, but complicated disorder of movement in which paresis and incoordination are variously blended, and which select by well-marked preference those muscles which are the readiest exponents of emotion. And as to the heart, in place of disturbed action and rhythm there is the proper emotional response with which everyone is familiar in his own person, an acceleration, namely, and often knocking impulse as well.

[1] Ziemssen's suggestion as to "the influence of restless muscular action upon aortic pressure and the working of the heart" (loc. cit. p. 437), fails to apply, I am convinced by repeated observation, in explanation either of the heart's irregularity or of its rate. It is indeed at once negatived by the admission that the heart disturbance of chorea is in no direct proportion to the violence of the muscular disorder.

Now at both the periods we are now considering, in childhood and in youth, both with cardiac irregularity and cardiac acceleration, we have, or we may have, apex murmur. It has been shown already that for such murmur no explanation is applicable except the functional one. And indeed, in the case of choreic patients, at that time of life when the so-called anæmic murmur is common, I know not how it is to be determined whether valve defect is due to chorea or to anæmia. Perhaps the same causes may operate in both cases, and the explanation of Dr. Nixon of faulty adjustment of valve and ventricle may serve for both. But, apart from these, there are the children to be considered. And, if we are still to apply the argument that the heart symptoms of chorea are in constant harmony with the rest, it is necessary to remember that there is another element in chorea besides those that have been alluded to. I mean *paresis*.

The real character of this choreic paresis has been somewhat obscured, I venture to think, by the prominence that has been given to those examples of it which have chanced to affect one side of the body only, and thus to give promise of material for illustrating the morbid anatomy of chorea. I would only remind the reader in this place as pertinent to the present inquiry, that a strictly localised muscular paresis very often mixes with choreic restlessness in children; that it is often very sudden both in its access and departure, giving to the affection an aspect of deformity which of itself it has not; that it may be recognised especially in a droop of the wrist or a falling of one shoulder, or feebleness of grasp of one hand; that it selects this part or that according to no known rule, and may in some instances show so prominently as to give the patient the appearance of paralysis rather than of chorea. Now all that can be said of choreic paresis as we see it in the limbs may be said as well of choreic murmur as we hear it at the heart. Both are characteristic of childish or at least youthful chorea, both are sudden in their development, variable in degree, and apt to come and go without any other corresponding change in the character of the disorder.

If then weakness and relaxation of the papillary muscles be

admitted as a possible cause of mitral regurgitation, and if a debility of this kind may arise temporarily from tobacco smoking or nervous exhaustion, much more should it arise in chorea where, apart from muscular fatigue, there is a special liability to paresis extending, as it would seem, to all the muscles which the disease is able to affect. And observe that this relaxation on the part of the papillary muscles, which is common both to anæmia and chorea, and productive of regurgitation in both, is not of the same origin in the two cases, nor always accompanied by precisely the same signs. In anæmia it gives rise to an equable murmur which only ceases with the gradual recovery of the patient; but in chorea it is (or it often is) a variable and inconstant murmur which hardly repeats itself precisely for two consecutive beats, and which appears and disappears without apparent cause, as does the similar paresis of the voluntary muscles, with which indeed, as I have repeatedly noticed, it may be strictly synchronous both in its time of arrival and duration. Moreover, with a murmur having this origin, we are rid of the difficulty which attaches to the doctrine of a fatigue paresis, and would require that the valvular defect should be in direct relation to the duration and severity of the chorea, which would associate it therefore, not so much with childhood, as with that violent form of chorea which is commonest at a later period of life.

Yet still the old objection will recur. Whence comes it that a temporary paresis of the valve apparatus is found to be associated after death, if not with endocarditis, at all events with a deposit of recent lymph on the edges of the mitral valve? That this appearance does not represent a true inflammation of the endocardium is rendered probable by its bead-like arrangement and limitation to the margin rather than the auricular surface of the valve. There is never ulceration, and where time has been allowed to elapse between the chorea and death, the valve is found to be in a normal condition. Only in cases where death supervenes either in the course of chorea or very shortly afterwards is this appearance met with, and in these not always. There is ample clinical and post-mortem evidence that it is neither lasting

nor injurious.[1] Mindful of this strict limitation of the facts to be interpreted, and of the necessity in the last resort of reaching a solution upon the principle of exclusion, the question suggests itself whether an imperfect closure of the mitral orifice, or rather such uncertain action of the valve segments as closes it at one time but not at another, might not, at the approach of death, and when with the flagging action of the heart fibrine is very readily deposited, so determine the seat and method of such deposit, as to produce at the edge of the valve the bead-like appearance that we see; the condition which immediately precedes death being one of the essential factors of the occurrence.[2]

It may be said, indeed, that whatever is true of the dynamic murmur of chorea must apply equally to the same murmur in anæmia and elsewhere. But of the post-mortem appearances of simple anæmia we know absolutely nothing. There is, however, a disease having a striking likeness to chorea in respect of its preference for the female sex, near connection with hysteria and with mental emotion, and apparently anomalous blending of functional with organic disturbance. In exophthalmic goitre we have at first acceleration of the heart and afterwards mitral murmur, a murmur which sometimes wholly disappears with the complete recovery of the patient, and sometimes is the precursor of organic mitral disease.

In conclusion, I would briefly recapitulate the facts which

[1] I venture to extract the following from a paper of my own, already referred to, on 'The Statistics of Fatal Chorea:'—

This post-mortem appearance occurs equally in those that die in connexion with chorea, and in those that die directly of it. Thus of Dr. Dickinson's 22, there are, as I reckon, 12 dying *with* but not *of* chorea; of these 10 have mitral valve vegetations and 2 have not. Of the 10 dying *of* chorea, 7 have vegetations and 3 have not. In Dr. Peacock's 3 fatal cases, all dying *of* chorea, 2 have vegetations and 1 has not. Out of 5 cases, the only examples I can find of fatal chorea with perfectly healthy heart, all save one, an old woman, are typical instances of death in and by chorea.

[2] The suggestion in the text is in fact that of Dr. Dickinson. "The beads," he writes, "are usually confined to the inner surface of the mitral valve and arranged along the attachment of the thin edge, where a line of minute but abrupt prominences is presented to retrograde blood, but an arrangement of more gradual slopes to blood flowing normally. Thus possibly the collection of fibrine is the consequence, not the cause, of the regurgitation." ('Pathology of Chorea,' loc. cit. p. 37.)

it has been the object of this paper to establish, together with the hypothesis which they seem to support. The facts are that in chorea the heart is apt to sympathise with the voluntary muscles at all ages up to the adult period, this sympathy being shown as well by dynamic murmur as by accelerated action, unevenness of rhythm, and, not seldom, the excited impulse common in hysteria, the particular manner of response being dependent upon the age of the patient. The hypothesis is that these several modes of heart affection correspond with as many modifications of chorea, which are exhibited not in the heart only, but in the voluntary muscles as well; these several regions sharing jointly, each in its own degree and after its own manner, in a disorder the area of whose influence is co-extensive with that of ordinary emotional disturbance: and, particularly that in all such variations the motor element of the affection is represented mainly by irregularity and unevenness of cardiac rhythm, the emotion element by acceleration, and the paresis element by dynamic murmur.

VISIBLE MUSCULAR CONDITIONS AS EXPRESSIVE OF STATES OF THE BRAIN AND NERVE CENTRES.

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(Continued from page 495, Part XII.)

SOME characteristic passive positions of the hand were described in the last paper as types; one other position must now be referred to—the "ortho-extended hand." Here the axis of the metacarpal bones and of the digits are in the same plane with the bones of the forearm. This is the position in which a strong man naturally holds out his hand when requested to put it forward. This may be considered as the antithesis of the "hand at rest." It was seen in Case V. *a*, presently to be described, in the left hand, when he held out his hands, while the right (paralysed) hand assumed the "nervous position."

A healthy child when it runs in its play, or to meet a friend, commonly holds out its arms, the hands assuming the "energetic position"; and this, as the result of observation, is I think characteristic of a healthy nerve-muscular condition; healthful children in pictures by the best masters are often thus represented. When a child is in the state of convulsion, it is well known that usually the hand is closed with the thumb turned in upon the palm, the fingers being flexed around it. Such a position of the hand in a child when ill is very indicative of a state of the nervous system predisposed to convulsions.

CASE X.—A child two years of age was admitted into Buxton Ward at the London Hospital, having had strong convulsions the day before. The child looked very ill, its

eyes were sunken, the body generally was rigid. The position of both hands was alike; the wrist was slightly extended, the bones of the thumb ortho-extended, or slightly more extended from the force with which its tip pressed against the last node of the index-finger. The metacarpo-phalangeal joints of the index and middle fingers slightly and equally flexed, the internodes remaining ortho-extended. The ring-finger rather more flexed than the middle one, separated from it, and the nodes slightly flexed. The little finger was in contact with the ring-finger, and assumed a similar position. I took a cast of the right hand, and the description at the same time. In a piece of sculpture exhibited at the Royal Academy two years ago a child's hands were represented so nearly in this character as to be very suggestive that the child was going off into fits.

The following cases may help to illustrate the advantage of studying common positions of the hand, that we may be able to detect others characteristic of weakness or disease.

Case V. *a*.—The notes of William D. have been already partially given; I now have to speak of the condition of his upper extremities. It has been said that he had been attacked with right hemiplegia; after a few months' treatment the condition of the arm improved, and the grasp of the hand became fairly good. In holding out both hands the left was straight with the forearm (ortho-extended), and the thumb straight with the fingers. In the right hand the wrist drooped a little, with slight extension of the metacarpo-phalangeal joints, and the thumb was extended—the nervous hand. There were some irregular and involuntary finger movements; these were chiefly flexor-extensor, occasionally slight abductor-adductor movements intervened, separating the digits. The muscular sense of this extremity was more interfered with than its muscular power.

Hemiplegia: involuntary movements of arm and face.

Case XI.—Arthur T., aged 10 years, came under my observation, May 1880, as the subject of a chronic left hemiplegic affection. His general health had been good, and the only

probable cause of brain disease was found in the fact that his father is syphilitic. His left upper extremity was wanting in muscular voluntary power and in muscular sense; certain involuntary movements of the hands and of the fingers were also observed. I now collect the notes describing the nerve-muscular condition of the hand. The left hand, when held out, assumed on the average the "nervous position"; not so the right, which was natural. A certain amount of involuntary movement of the fingers was seen; the middle finger moved the most; during the examination, and at other times when his fingers were curling up against his will, he would frequently use the right hand to straighten the fingers of the left. As to the kind of movement, it certainly was not voluntary, and on several occasions a similar movement was seen repeated. The dynamometer showed the power of the right hand as 47, against 36·5 on the left side.

I had often noticed that the boy looked much distressed when I examined the left arm, and this appeared not surprising considering that, with good bodily health, he was losing the use of the left arm, and at the same time there was family trouble from the father being out of work. Looking at the lad's face, one observed distinct over-muscular action in the upper zone, causing in the middle of the forehead transverse and vertical furrows, an appearance commonly resulting from the condition of brain associated with grief. For a long time I was deceived by this face, and thought the boy depressed, but later, on cross-questioning him, and inquiring of his mother, he appeared not only unconscious of these movements, but not to feel any mental distress. I was therefore compelled to regard the facial movements, which were suggestive of the expression of distress, as analogous to those of the fingers, which more resemble athetosis than any other phase of involuntary movement. The movements of the fingers were slow, involuntary, and unconscious; no child in chorea puts up his hand to straighten out the fingers which have curled up on the other hand. There were no general twitching movements, and those movements that did exist were not twitching in character. These points appear to indicate an analogy to athetosis, rather than to chorea, both in the hand and in the face.

CASE XII.—*Hemiplegia, malingering.* — Charles Giles, a country-looking man, aged 29 years, was an in-patient at the London Hospital, December 1880. He was admitted for right hemiplegia with anæsthesia. Suspecting the man a malingerer, I carefully noted the following points. "The right upper extremity is held out well and firmly, on a level with the shoulder; it is apparently easily kept out, and well sustained, as no muscular tremor or twitching is seen. The hand droops vertically from the wrist, the joint being flexed at about a right angle, and all the digits in a straight line with the metacarpal bones." This drooping of the wrist, as if hanging dead at the end of an active arm, is a condition totally different from the more or less flexed position usually seen in hemiplegia. He represented an arm without tone in the extensors of the hand and digits, and without rigidity. The hemianæsthesia was easily disproved; the electro-contractility of the two extremities was equal; he was sometimes caught using the right hand, and finally ran away, having inquired as to the locality of another hospital.

Before passing away from the consideration of the upper extremity as an index of brain conditions, we must notice the more active conditions of muscular movement. Equally characteristic with the passive positions of the hands are the muscular movements of the fingers,—twitching, tremors, and rhythmical movements. These conditions have been more fully studied and described than the characteristic passive positions (postures), and they have long been considered as visible muscular conditions expressive of the states of the nerve centres. The varieties of finger-twitching may be described as:—

(1.) *Flexor-extensor;* the primary movement being that of flexion, followed by a secondary extensor movement. This may be seen in a variety of cases, and in particular is seen in what is called "picking the bedclothes" in the typhoid state preceding fully developed coma.

(2.) *Extensor-flexor;* the primary movement being that of extension, followed by a secondary flexor movement. This is common in the slighter forms of chorea and in nervous

children; such twitches usually constitute the subsultus tendinum so indicative of exhaustion in the course of typhoid fever.

(3.) *Abductor-adductor* twitches; the movements consisting in lateral separations of the fingers, followed by their being drawn together again.

As to "finger-twitching," the "nervous hand," and "the relaxed orbicularis oculi," the following statistics are from an analysis of 34 cases from my note-books of the East London Children's Hospital:—

Finger-twitchings in 19.—Twitchings alone	in	8	cases.
With the nervous hand . . .	„	8	„
With relaxed orbicularis . .	„	3	„
		19	„
The nervous hand in 19.—Nervous hand alone	„	7	„
With twitchings	„	8	„
With relaxed orbicularis . .	„	4	„
		19	„
Orbicularis relaxed in 10.—Orbicularis relaxed alone . .	„	3	„
With the nervous hand . . .	„	4	„
With twitchings	„	3	„
		10	„

As to the general character of this group of 34 nervous cases in which nerve-muscular signs were specially noted, no cases of known organic brain disease were included, and all were under 15 years of age. I have abstracted and summarised the diagnosis of the 19 cases in which the "nervous hand" was seen:—"Headaches," 6; "Neurotic temperament," 3; "Anæmia and headaches," 2; "Headache and somnambulism," 1; "Restless sleep," 1; "Laryngismus," 1; "A dull child with congenital ptosis," 1; "Old rickets," 1; "Debility," 2; "Slight chorea," 1. The cases of "finger-twitching" had the same general characters as those with the "nervous hand," therefore I do not further describe them.

As to the kinds of finger-twitching, the varieties were noted as follows:—Simple twitching, 9; flexor, and abductor-adductor, 5; flexor, 3; abductor-adductor, 1; extensor, and

abductor-adductor, 1. As shown in the tables above, the "nervous hand" was associated with "finger-twitches" in 8 cases.

In cases where the right and left hands were compared, we find a difference in 6 cases, always to the disadvantage of the left hand; it specially presented "the nervous position" in 4 cases, and finger-twitches were specially marked on the left side in 2 cases.

The cases with "relaxed orbicularis" were specially marked by recurrent headaches, some with optical illusions and scarlet zigzag forms.

In 2 cases herpes zoster occurred while under observation.

The various positions, and movements of the head upon the spine, now claim our attention in as far as they depend upon and indicate the condition of the nervous system.

If we describe two axes of the head: (1) Interparietal, passing from one side to the other above the ears; (2) Occipito-frontal, passing from the occipital protuberance behind to the centre of the forehead in front, we can by referring to the positions of the axes define all the positions and movements of the head.

A. *Flexion and extension*—i.e. bending forwards and backwards of the head. Here the occipito-frontal axis has its anterior or posterior extremity depressed, but there is no deviation to either side, and the two ears remain at the same level.

B. *Rotation*—i.e. rotation of the occipito-frontal axis in the horizontal plane, the head remaining erect, and the interparietal axis horizontal, without flexion or extension.

C. *Inclination*—i.e. depression of one or other extremity of the interparietal axis, in which case one ear is lower than the other. Inclination may occur without either flexion or rotation, but is commonly associated with both. Right inclination means depression of the right extremity of the interparietal axis; left inclination, depression of the left extremity.

By means of these three terms we can, I believe, define all the possible positions and movements of the head. No account

is here given of the alterations of the positions of the head due to movements of the cervical spine. Flexion and extension of the head are of frequent necessity and physiological import; rotation is frequently necessary to move the eyes towards an object looked at, or to direct the ear towards a source of sound.

Inclination is, I think, often of other significance; it often indicates weakness, it is more often seen in young girls and weakly persons than in strong men. Inclination, with rotation to the same side, with slight flexion, is a position of the head very commonly seen in choreic girls, and then often accompanies an awkward ill-balanced position of the spine.

Inclination of the head, with rotation to the opposite side, and slight extension, is indicated by Sir Charles Bell in his figure in Adoration. In seeking general information as to the signs of cerebral adequacy in an infant, that is, whether the infant has a good sound brain, probably capable of due development, we always notice whether it can hold its head up, whether it usually keeps it erect when sitting up. In drawings of babies, we often see them represented as weak, evidently incapable of holding the head erect, the artist representing the want of strength of infancy by personal weakness and debility; this may be seen on comparing certain modern paintings with some of Raphael's infants.

An example of abnormal position of the head is the extreme retraction often met with in infants in brain disturbance, and also in cases of epidemic cerebro-spinal meningitis in adults. In all cases it depends upon tonic spasm of the extensors of the head, and is of grave import as indicating brain disease.

Case XIII.—January 1879. George W., age 4 months; complaint was made that he was stiff and unable to move his head. Fourteen days previously he had been taken ill suddenly with slight feverishness, and in a few days he could not sit up, and he fell into the condition in which he was when seen. He could move his arms and legs, but his head was distinctly retracted, and any movement of the head caused evident pain; there was no tonic spasm except in the muscles which retract the head; he looked intelligent, and the abdomen was natural. The tonic spasm lessened in a

week and passed away in three weeks; he remained two months under observation and recovered completely.

Such cases of retracted head are not uncommon, I think most of them die; it seems certain that in all cases the spasm depends upon nerve stimulus.

A curious and, as far as I know, unexplained chronic movement of the head in flexion and extension is occasionally seen in infants; the head constantly nodding up and down—a similar rotatory movement sometimes occurs.

Case XIV.—Elizabeth C., age 11 months, appeared healthy and well, and there was no complaint made except as to the head movements. These movements had commenced about a month previous to the time of observation; at first the mother did not notice them much, and there were never any signs of pain or general disturbance, the limbs were in no way affected. The infant looked healthy and well: the head-movements were simply rotatory in character; they came on, lasting a few seconds, then passed off again, leaving the head quiet, with the power of natural movement; then again three or four lateral oscillations succeeded, apparently never any vertical ones. There were never any general convulsions; no cause was assignable, and there was no history of injury or exposure to cold. The case came under my care at the Children's Hospital, Birmingham, in 1876.

Case XV.—Margaret McC., aged 8 months, seen April 1, 1879. She was brought under observation on account of constant nodding movements of the head. The movements were simply in the vertical direction, extension and flexion, being a series of nodding movements; these had continued for three months. The head was not retracted, the sutures appeared normal; the ribs were slightly bended. General health was good. The child continued under observation a month, the head-movements remaining unaffected; light appeared to increase the movements. These movements appeared somewhat analogous to those of nystagmus.

In cases of cerebral hæmorrhage, softening, tumour, and other coarse brain-disease, paralysis of one or more cranial nerves often forms a marked diagnostic symptom. As has already been said, facial paralysis from brain disease presents

certain characters distinguishing it from a lesion of the facial nerve. Ptosis and strabismus are not uncommon results of coarse brain disease; these facts teach us to look to the condition of the muscles supplied by the cranial nerves when examining the condition of the brain.

The first two cranial nerves are purely sensory, having no motor function; the third, fourth, and sixth supply the eye-muscles; of the movements of the eyes as indicating brain conditions much has been said already. The seventh nerve supplies the facial muscles, and its action has been discussed. There remain then for consideration the motor division of the fifth nerve (masticatory), the eighth, and the ninth.

Fifth nerve.—Tooth-grinding is produced by the action of the deeply-situated pterygoid muscles; champing of the jaws and trismus are produced by the masseter and temporal muscles; all these muscles are supplied by the fifth nerve, and it is to their condition that we must look for information as to the condition of the central origin of the nerve. Tooth-grinding when it has become a habit is indicated by the flattened condition of the tips or edges of the teeth which may be ground down—a sign that may be particularly seen in the incisors and canines. Ground teeth are very common in nervous children, such as those who suffer from recurrent headaches, restless sleep, somnambulism and finger-twitching. In lunatic asylums and wards for imbeciles it is very common to hear tooth-grinding on every side; in such cases tooth-grinding is a sign of central irritation of the fifth nerve; it is well to bear in mind that the sensory branches of this nerve supply the membranes of the brain and the external parts of the head.

Trismus is tonic contraction of the masseter and temporal muscles which close the jaws, and it is a common symptom in tetanus, epilepsy, and hysteria. Seeing that the slight disturbances occurring during sleep in many children caused the pterygoids to contract rhythmically, it is not surprising that grave disease should cause spasm of the other muscles supplied by the fifth nerve.

The ninth nerve is motor to the tongue, and this organ being a mass of muscular fibres running in various directions almost unsupported by bones, is very sensitive to changes in

the nerve-centres. In chorea the tongue is often jerked in and out in a manner quite characteristic of the disease; in other cases it is easily kept protruded, and its substance is seen to be in a condition of constant movement. Such irregular movement is very common in nervous children; a tremulous tongue is characteristic of alcoholism, and general paralysis of the insane. In hooping-cough we see tonic spasm of the tongue causing its violent protrusion, which is frequently so strong as to cut the frænum by stretching it over the lower incisors; the centre of the tongue is depressed, while its margins and tip are raised. Defective speech is not necessarily a lesion of the muscle of the tongue; but we can only know the condition of the centre of speech through its power to guide the muscles of the tongue and others used in speech.

Concerning the eighth pair of nerves, the distribution is partly to voluntary muscles, but the pneumogastric is distributed to many organs, and largely to organic muscular fibres; this fact brings us to the consideration that the tone or action of organic muscular tissue may indicate states of the nerve-centres. As the examples to be quoted are not cases of the visible action of muscles, they will only be enumerated. Palpitation may occur without disease of the heart, the action may be irregular, as the result of chorea or of coarse brain disease; the controlling action of the nerve over many glands and organs is probably due to its action upon the muscular walls of the vessels; there is also evidence that in asthma, bronchial spasm is due to irritation of the pneumogastric; in functional aphasia, the recurrent branches do not convey the due stimulus.

It is a matter of very common experience that children and adult patients "hold themselves awkwardly;" stoop, or otherwise give the spine and trunk an ill-balanced position, due to want of nerve-muscular energy, and characteristic of the condition of exhaustion and weakness. Doubtless there is much expression in a torso; in many cases a weakly condition is indicated by a stooping attitude, with a lolling over to one side. It is difficult to indicate in precise terms the positions and movements of the spine, and perhaps this is one reason why so little is known about the action of the muscles of the

back. In stooping, the spine may remain symmetrical, the bends of the column being only in the antero-posterior median plane, and not deviating laterally.

We may then distinguish symmetrical from asymmetrical positions and movements of the spine. Some further remarks will be made on this subject under the head of chorea.

As to the lower extremities, I have fewer observations to refer to than with the upper limbs. The gait and manner in walking may be characteristic of brain or cord disease. Conditions affecting the muscles of both lower extremities are usually dependent upon disease of the cord, and most of the signs by which spinal disease may be localised are derived from examination of the muscles of the legs. Cramp in the feet and legs is due to tonic spasm of the muscles, and probably is usually produced by overstraining of the muscles, or by indigestion, &c., i.e. it is not dependent directly upon the condition of the nerve-centres, and therefore is not to be taken as a direct index of their condition.

Reflex actions are used as means of localising and ascertaining the condition of nerve-centres. Here, again, visible muscular conditions are the indices; but on this subject no more will be said now, as it hardly comes within the scope of this paper.

As to the proximate cause of the passive positions or postures of extremities—the hand, the spine, the face, &c.—it is the relative tone of the muscles involved that brings this about; mainly the relative tone of the flexors and extensors; and this relative condition of tonicity results, it is believed, from the condition of activity of the nerve-centres corresponding. Hence it is probable that in observations of the involuntary postures, or passive positions of the limbs, hands and digits, we may find indices which we may record in cases indicating the relative tonicity of groups and sets of muscles, and the conditions of the corresponding nerve-centres.

No doubt the position of the limbs is simply the position of the bones, and this is simply the result of the relative activity of the muscles acting upon them; the action of muscles is the cause of the posture or movement, and the posture or movement indicate the action of the muscles, and so likewise the

action of the nerve-centres corresponding; therefore the postures may indicate the state of nerve-centres.

In describing postures it is probably worth while to attempt greater accuracy by indicating the angles of flexion and extension; adduction or abduction in the different joints. The condition of the hand in ortho-extension is that of a balance of the flexors and extensors; in the "hand at rest" the flexors have the best of it, and the amount of hypertonicity of the flexors may be indicated by giving the relative angles of flexion at the different joints, the less this angle the greater the hypertonicity of the flexors, i.e. the angle of flexion and the hypertonicity of the flexors are inversely proportional. In the ortho-extended hand there is no angle of flexion or extension.

The principle of antithesis is used by Darwin to explain certain forms of expression of the emotions, and I think that every physiologist must grant the muscular indications of emotion are due to the state of the nervous system, its structure and its activity. The dictum then involved in the assertion of this principle is that opposite conditions of muscular action indicate opposite states of the nerve-centres, and conversely, that opposite states of nerve-centres will cause opposite muscular conditions. The most natural opposite conditions of the nerve-centres are activity and inactivity, or activity and exhaustion. It is not only in the case of motor nerve-centres that the nerves become so exhausted that they cannot repeat their proper functions. The eye, after it has been fixed on one strong colour becomes incapable of perceiving that colour longer till it has been rested, but it may readily perceive the complementary colour; again, after having looked long at a bright light, a black spot only is seen when we look elsewhere. The following appear fair examples of antithetical postures.

1. The erect figure—the stooping figure.
2. The nervous hand—the energetic hand.
3. The hand at rest—the ortho-extended hand.
4. Orbicularis oculi relaxed—the orbicularis energised in laughter.

Chorea has in this essay been many times referred to,

because it is a condition of brain which we know only through the effect produced upon the muscles of the brain, and this condition affords abundant examples of various forms of nerve-muscular movement. It will then probably serve my purpose if I enumerate some of the principal muscles and groups of muscles that may be affected. In different cases very different groups of muscles may be affected, thus indicating the very different brain areas that may be choreic. It is suggested that in studying a case of chorea we should try and indicate the extent of brain affected by specially indicating the choreic area. The following points then should always be looked for —as present or absent in any case described—and the order of invasion of groups of muscles or their recovery should be observed:—

1. In examining a case to prove the fact of chorea it is very convenient first to look at one or both hands, held out free and disengaged. The kinds of movements of the hand and fingers have been dwelt upon and described.
2. The upper and lower extremities present the greatest mass of the choreic movements. It is important to note whether the finer or coarser movements be the most affected; the amount of involuntary movement, and the power of voluntary act that is left.
3. Hemiplegic varieties are common; the least mobile side may be much weakened, though not much moved.
4. The face. Varieties in this group of muscles have been discussed.
5. The soft palate may present marked movements of an irregular twitching kind, the levator-palati muscles working distinctly. I do not refer here to the dragging of the palate by the choreic twitchings of the tongue, but to the primary twitching of the palatine muscles. In some cases the levators are distinctly seen twitching upwards. This symptom is often absent in chorea, and when seen, I have observed that it has usually passed off early.
6. *Tongue* may be markedly jerked in and out. When

protruded, it may present much movement, but still be kept out a fairly long time.

7. Eyes. Upper eyelids often strongly retracted.
8. Head in the active stage is often moved much. During convalescence, and when the active movements have passed off, a lolling of the head to one side is common; i. e. inclination with rotation to the same side, combined with slight flexion.
9. Spinal muscles and trunk are often affected, but how to describe their condition I do not know. The child often balances itself very ill, throwing the scapular and upper dorsal region too far back, and thrusting the pelvis too forward, the spine still remaining symmetrical. It has sometimes seemed to me that the awkward appearance was due to want of adaptability of the proper compensations in the movements of different muscles.
10. The respiratory muscles. These may be affected much or little; the alæ nasi muscles may be affected also.
11. The vascular centres. The heart's action is sometimes irregular.
12. Varieties.—Hemiplegic.
    Paralytic.
    Local chorea; in Nerves IX., V., &c.

The significance of the action of muscles as indicating brain conditions has long been dwelt upon by writers. Camper, who wrote in 1821, has shown how the Laocoon presents evidence as to how deeply the ancients had investigated the influence of pain as expressed in the figure and the muscles. In this group, "not merely does the face, but the arms, legs—in short, all the muscles of the body indicate anguish." Further on he quotes from the words of Paulo Somazzo's work, 'Dell' Arte della Pittura,' published 1531, in which he describes the influence of the passions upon the muscles of the face, and still more minutely the different postures and contortions of the body. Camper there complains that authors have usually either confined themselves to appearances, or have "reasoned metaphysically concerning the operations of the mind, without

attending to the physical causes of the changes produced by these operations, but in my opinion (that is, Camper's) speculations concerning the manner of the soul's working, or concerning the seat of the soul, are of no use to the artist. These belong to metaphysicians, who, by the way, lose themselves in a labyrinth of terms, or words with no definite meaning, without having in the least explained the action of this immortal principle upon the corporeal and mortal frame." Camper then proceeds to give examples of the conditions of the muscles as indicating conditions of the mind, and then says, "the observation deducible from these effects is, that in every emotion of the mind particular nerves are affected; consequently every painter ought to make himself acquainted with the construction and connection of the nerves productive of these changes."

John Bulwer in 1649 published his work entitled 'Significative Muscles of the Affections of the Mind.' He expresses his opinion that every motion of the mind is indicated by a corresponding motion of the muscles. This is the same idea as Camper expressed later, and as is now well known to physiologists, that all postures and movements are the result of changes in nerve-centres. To study the conditions of the mind it is necessary now, as in former times, that the positions and movements of the body should be largely observed. Bulwer says, "When we assent, affirme, yield, grant, vote, confirme, confesse, admit, allow, or approve of a thing, &c., we are wont to nod or bend our head forward, the naturall reason of which motion in these, seems an approving, which is made by the Imagination, seeing, or hearing, somewhat done or said which accordeth very well, and this power remaineth in the Braine or forehead part of the head where in the cell and Seat of the Imaginations lieth. When any of these things give it contentment, suddenly it moveth the same, and after it all the muscles of the Body." In all cases, after describing the expression of a state of mind, Bulwer endeavours to explain which muscles take part in the act.

If anything can aid our studies of man, the matter becomes of interest to several classes of writers, to all those who study the body of man as indicating the activities of this brain or

mind, and as giving the knowledge of the means whereby the idea of his mental states and feelings may be expressed. If the matters discussed in this essay are of use in this direction, they concern not only the physician, but also the artist.

It is the work of the painter and sculptor to express by form the conceptions he may conceive of the condition of men and women in certain conditions of mind, states of strength and weakness; expressions of mental and physical pain, states of rest or repose, feminine coyness and defiance; the poet has to describe all these things in words. Clearly, also, it is a mistake for conditions of the limbs characteristic of disease to be used in art as mere expressions of feeling, unless the feeling be the result of disease.

There is then in the subject before us, "the visible conditions of the muscles as expressive of the states of the nervous system," a field for observation and description in which the artist and the physician may work together, observing and analysing with as much exactness as may be, the mode by which the varying conditions of the brain and mind are indicated to our eye, and may therefore be described by words, or by drawing or sculpture. We must study man in all aspects of the case, and when we see in the face, limbs, or body indications of his brain or mental condition, we should analyse and describe—first, the position of features and parts as we see them, then the muscles which produce these positions or movements, knowing that the muscular condition which has produced the movements or positions is the result of the state of the corresponding nerve-centres. It has been said that a man's face is the index of his mind, and this is true, for all the varying changes of expression in the face (except those of colour) are due to changes in the facial muscles, and these solely depend upon changes in nerve-cells.

The knowledge that we already possess of the nerve-centres is from observation of the condition of the muscles. In a given case, comparing the state of the muscles during life as they may be affected with paralysis or spasm with the brain lesion found after death, and by collecting and comparing many cases, it has been found that destructive or irritative lesions of certain parts of the brain cause paralysis or spasm of a certain

set of muscles corresponding. It is probable then that by carefully continuing these methods of examination—that is, by describing with accuracy all states which are indicated by conditions of the muscles—we may add still more to our knowledge of the functions of different parts of the brain, and gain a further insight into the pathology of that large group of nervous centres termed "functional." And to conduct such inquiries is a proper work for the physician. The objects of this paper have been to stimulate observations, and to direct and guide inquiries by definite lines and methods of investigation. Descriptions and definitions of some characteristic positions of parts and conditions of muscles have been given to aid in the clinical record of cases. Practical points are to some extent insisted on as specially characteristic means by which we may judge of the condition of the nervous system or of some centres forming it.

Irregular muscular movements, such as different forms of spasm, tremors, tic, &c., have often attracted much attention; but of these nerve-muscular conditions we have nothing more to say here. When dealing with a nervous case, however obscure and difficult to describe, if there be any such spasms or tremors, &c., local paralyses or weakness, movements or special passive positions, the case may be characterised by the description of these signs, and such indications of the state of nerve-centres are worthy of clinical study.

AN ELECTROTHERAPEUTICAL SUPERSTITION: "THE GALVANISATION OF THE SYMPATHETIC."

BY A. DE WATTEVILLE.

UNDER the title of "Galvanisation of the Sympathetic" has been, and is being, accumulated a large amount of that kind of literature which has done so much to cast discredit upon the subject of electrotherapeutics. Erroneous physics, imaginary physiology, fantastic pathology are there worked by processes unknown to ordinary logic into a sort of mystical creed. This heirloom of Remak's Benedikt[1] of Vienna claims, in accents of which the passionate enthusiasm is only equalled by the absurdity of the arguments he used to defend it. The mantle of the prophet has fallen upon his shoulders! Neftel[2] of New York (translating Benedikt without acknowledgment) pronounces the talisman to be one of the greatest therapeutic discoveries of the century. Speaking of "the rigid form of Infantile paralysis," the author of "How to use a Galvanic Battery" quaintly remarks, that "if these cases depend upon adhesions or exudations in the medulla, their absorption may possibly be promoted by localising a voltaic current in the superior cervical ganglion of the sympathetic. There seems no doubt that such an application causes a dilatation of the blood-vessels at the base of the brain, and is likely therefore to promote absorption."

Not only the cervical, but the abdominal sympathetic, the splanchnic nerve[3] and plexuses are each to be singled out by

[1] 'Elektrotherapie,' 1st ed., 1868. 2nd ed., 1874.

[2] 'Galvanotherapeutics,' 1878.

[3] The following observation is adduced as proof positive "that with the galvanic current we are able to exercise a direct influence on the splanchnic

the current in order to display its magic powers for cure. All the known, and many imaginary, diseases of the encephalon are to be relieved by acting on the superior cervical ganglion —and, let us not forget, "the *negative* pole on the ganglion, the positive on the nape of the neck is the really curative arrangement."

Galvanisation of the sympathetic is likewise a specific in a very respectable number of spinal complaints, rheumatic gout, optic neuritis, spasms in every part of the body, &c. And how could it well be otherwise? For, as Neftel profoundly observes, "by galvanisation of the cervical sympathetic we may produce hyperæmia or anæmia in the vasomotor centre"—a process, by the way, analogous to that of a dog running after his own tail—and further, "in the inhibitory centre of reflex action of the spinal cord, in the convulsive centre, and consequently prevent or call forth epileptic attacks and other cerebral disorders." So much for the rationale of the process: we must all deplore, however, the absence of any explanation as to the comparative therapeutical uselessness of hyperæmia and anæmia in these very centres, produced by nitrite of amyl and ergot, for instance; and simply believe that vasomotor effects obtained by the galvanic agency possess some occult powers *sui generis* hitherto beyond our comprehension.

However extravagant the pretensions put forward by the galvanisers of the sympathetic, and puerile their attempts at giving an objective basis to their beliefs, it may be granted that their method of electrisation is often useful. We must not allow ourselves to shut our eyes to clinical facts simply because presented to us in an absurd language.[1] Still less

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nerves." Neftel applies a continuous current of fifteen cells from the back to the prostatic part of the urethra: "in less than half a minute the surface of the body assumed a deathlike pallor from the tetanic contraction of the small arteries, and this was followed instantly by syncope."

[1] Professor Erb's sober remarks stand in refreshing contrast with the tone we are accustomed to find adopted. In his experience good results have been obtained, in several spinal affections, from galvanisation with one pole over the vertebræ; the other in the subaural position. So far from calling to his aid any problematical sympathetic or vasomotor effects to explain such results, he merely hints "that in this way, possibly, a better permeation of the cord by the current is secured."

should the more general interests of electrotherapeutics suffer from such a cause. But it behoves every one whose name is connected with this branch of medicine to utter no uncertain sound on this subject; and at the risk of incurring the charge of aggressiveness, ruthlessly expose the pseudo-physiological inanities which have so long been a grievous stumbling-block in the way of common-sense and true progress.

The first point we must define is how much of the current we use actually passes through the cervical ganglion. To determine this with absolute accuracy is of course impossible; but a knowledge of the ordinary laws of electrical diffusion in conductors is sufficient to enable us to form some idea on this subject. Some writers have gone so far as to deny the possibility of reaching the sympathetic at all with our currents; a statement which need only be adverted to here as an illustration of the singular want of physical knowledge, even among men of high standing in the medical profession.[1] Erb, Burkhardt and others have, besides, put themselves at the unnecessary trouble of demonstrating the conductivity of the sympathetic by galvanometric experiments.

The factors regulating the density of a given current through a given point of the human body are several: first, the surface of the electrode; second, the distance of the electrode from the structure to be influenced; third, the relative conductivity of the several tissues; lastly, the position of the second electrode.

In the process known as the galvanisation of the sympathetic,[2] one of the poles is placed below the ear over the seat of the superior cervical ganglion. It should be of small size, so as to obtain the greatest possible density of current. We cannot, however, prevent an enormous diffusion of the current to take place immediately beneath the integuments. The ganglion is somewhat deeply placed, and surrounded by

[1] The same view has been held with reference to the bony envelope of the brain spinal cord. How living animal tissues, soaked with a saline solution, could be thought to be insulators is difficult to conceive.

[2] Fischer has proposed to call it "of the neck," a name that has the merit of involving no theory, but is rather vague. I should prefer a designation implying merely the position of the electrode: *subaural* galvanisation is perhaps as convenient as any other.

muscles and blood-vessels—all better conductors than itself, so that but a very trifling proportion of the electricity reaches it. To this we may add here, by anticipation, that weak currents only are safe in this region, owing to their ready diffusion through the base of the brain, and the very unpleasant symptoms thereby produced.

The only position of the second electrode by which any notable concentration of the current could be secured is inside the pharyngeal cavity, as proposed by Gerhardt. But to my knowledge all the other observers place that electrode at the nape of the neck or at the manubrium sterni. Now this is, as far as the superior ganglion is concerned, absolutely as if the application was strictly unipolar, and the "indifferent" electrode applied to some distant part of the body. In other words, the current as soon as it reaches the better-conducting tissues under the integuments diffuses in *all* directions; numberless paths are opened before it, through each of which it circulates in quantities inversely proportional to their resistance. Now this resistance in each case depends upon two factors: the diameter (or sectional surface) of the path, and the specific resistance of the tissues which compose it. If we assume the latter to be a constant mean, it is obvious that the imaginary direct path, the sectional surface of which is represented by the outline of the sympathetic nerve, is very small compared with the aggregate of the remaining paths through which the current circulates.

Among the authors who have treated the subject before us, two entirely different ideas are constantly found intermixed by a sort of mental jugglery which is apt to deceive the unthinking reader into acquiescence. In discussing the curative value of the galvanisation of the sympathetic, we must draw the same strict distinction between the immediate ends we have in view, as when we consider the curative influence of any other therapeutical agent. Do we intend to act directly upon a morbid condition of the nerve itself; or do we intend to stimulate the functions of the nerve, and so indirectly influence the condition of some other organ? In the first instance, it is obvious that the weak currents that reach the nerve may perfectly be entitled to the possession of certain

therapeutical properties—exactly as small doses of many medicines do doubtlessly possess. But if on the other hand we pretend to obtain our object by calling forth the physiological activities of the nerve, we must use quantities considerable enough to obtain clear proofs of such an action. Now in the vast majority of cases galvanisation of the sympathetic is claimed by its advocates to act in the latter way, that is indirectly by inducing changes in more or less distant organs, such as the brain, the eyes, and other parts. Disease of the sympathetic itself is comparatively rare and usually marked by distinctive symptoms; this part of the subject is, however, beyond our present scope. The questions before us are therefore: What are the effects of a physiological stimulation of the sympathetic? Do we obtain such effects in the medical application of electricity to this nerve in the living man? Our answers need not occupy many words. Of the two chief functions of the cervical sympathetic, which are to constrict the vessels of the head and dilate the pupil, we observe neither the one nor the other in practice. The insignificant and even contradictory results of numerous observations on the effects of subaural galvanisation upon the pupil cannot be appealed to; for it is notorious that the excitation of any sensory nerve, such as is necessarily involved in percutaneous galvanisation, is enough to produce some dilatation of the pupil. Further, if it could be shown that the effects observed were due to the electrisation of the nerves it would but give a measure of the amount of vasomotor change produced at the same time in the brain. Now the most accurate observations with the pupilloscope are necessary to discover the faint dilatation that is said to occur. What sane being could then attempt to balance a whole scheme of therapeutics upon such a minute basis?

But again, strange to say, it is usually assumed that the effect obtained on the circulation of the brain is one of dilatation, not of constriction, of the arterioles. The curious hallucinations under which many electrotherapeutists labour on this as on other points have been well illustrated by the quotation above given from a noted expert in these matters. It is true that, on the ground of certain experiments, it has been held that the galvanic current applied to the nerve acted

differently from the faradic, which always causes contraction of the cerebral vessels. Onimus and Legros[1] say that the ascending current contracts, whilst the descending dilates the arterioles. But it must be remembered first that these authors hold peculiar views about the "peristaltic" action of the muscular coat of the vessels. Second, that accurate investigations by Professors Vulpian,[2] Chauveau,[3] Fischer,[4] and others have failed to confirm these results. Every kind of stimulation of the exposed nerve yields the same result, constriction of the blood-vessels; and percutaneous electrisation, in Vulpian's hands, has had no effect whatever upon the arterial tension. Chauveau has shown that descending currents produce rather more contraction than ascending currents, the explanation of the difference being plainly the relative position of the negative pole in the two experiments. Let us note here that mistakes have occurred as to the names "ascending and descending current" with reference to the sympathetic: its course being upwards, the descending current is necessarily the one in which the negative pole is nearest to the head. Two things are certain if, as alleged, any notable arterial dilatation occurred during subaural galvanisation: the first, that we should expect to find some evidence of it in the integuments; the second, that this effect would readily be explained by some reflex action from the centripetal filaments of the vagus or cervical cutaneous nerves.

Much labour has been expended over the investigation of the retinal vessels during galvanisation of the sympathetic. Here, at least, it was thought decisive evidence of the vasomotor effects of the process would be obtained. Beard and Rockwell devote a considerable space to the description of

[1] 'Traité d'Electricité Médicale.'

[2] 'Le Système Vasomoteur,' vol. i.

[3] Original communication in Teissier's 'De la valeur thérapeutique des courants continus.' (Paris, 1878.)

[4] Fischer ('Centralblatt,' 1878, p. 124) after many experiments has come to the conclusion that in order to obtain higher vasomotor effects, faradisation or galvanic interruptions must be employed in the case of the sympathetic. Prsewosky, however, has found that polarisation of mixed nerves had an effect on the temperature of the limb. With the negative pole it was slightly raised, with the positive slightly lowered; but I am not aware that these results have been since verified.

their results, which were contradictory.[1] No wonder since the vasomotor nerves of the retina do not pass through the cervical ganglion, but are contained mainly in the trigeminus! In all fairness it must be said that this is a fatal objection to the argument derived from such negative results against the possibility of reaching electrically the cervical ganglion in man.[2]

Among the curious traditional proofs of the value of the process faithfully preserved in electrotherapeutical treatises is the relation of one or two cases where the *arm* of the patient used to become covered with perspiration every time he was galvanised.[3] Apart from the curious localisation of this phenomenon, it is in addition precisely the opposite of what physiology would make us expect. The cervical sympathetic does not supply the arm, and its irritation should produce a diminution of the sudoral secretion. This leads us to consider a fact which is coolly ignored by most writers, that is the position of the other pole, which is usually applied to the nape of the neck at the level of the origin of the brachial plexus. This is surely quite sufficient to explain a great deal of the effects attributed to the galvanisation "of the sympathetic"! Indeed a great therapeutical importance is laid upon "the production of an- or katelectrotonus of the upper part of the cord" by some of the very staunchest believers in the peculiar efficacy of the subaural position of one of the electrodes. Another circumstance to be noted *en passant* is the absence of any definite rules given in books as to the side on which the sympathetic is to be galvanised in unilateral brain disease. For instance, it would appear that most electrotherapeutists apply the current on the same side as the paralysis, that is, "act on the cerebral circulation" of the sound half of the brain. Surely this is hardly rational, and can be only explained by a reference to the historical development of the question. Remak held that the sympathetic had most extensive functions, among which to keep up the muscular tonus

[1] "Much seemed to depend on the temperament and condition of the individual; what would cause contraction in one would in the other cause dilatation." And these inanities are gravely reproduced by other authors, with commentaries!

[2] See Buzzard, 'Practitioner,' Feb. 1873.

[3] Moritz Mayer, 'Die Electricität,' &c. Cf. Eulenburg, loc. cit. pp. 30, 31.

of the whole body.[1] Electrisation of the cervical sympathetic on the side of the paralysis was supposed, therefore, to act directly upon the paralysed limb. Later, when the discovery of the vasomotor system began to carry away pathological theories to the absurd length still fresh to our minds,[2] the rationale of the process was changed to suit the times, but the practice remained.

Remak himself started the theory of "indirect catalysis" in explanation of the curative influence of subaural galvanisation in certain cases. He supposed in several forms of brain disease that the lesions consisted in effusions or adhesive exudations which the vascular dilatation produced helped to remove—a method of argument, like the "post hoc," not rare in medical literature, and which consists in inferring the morbid condition from the very therapeutical hypothesis which it is supposed to explain, or *vice versâ*. The tradition has been but too faithfully kept up by the disciples, as we have already had the opportunity to point out. It is evident that in subaural galvanisation a large number of structures, each as fruitful a field for electrotherapeutic imaginings as the sympathetic itself, receive their share of the electrical influence. I have already mentioned that the effect of the second pole on the cervical portion of the cord is usually kept out of sight. But besides this, the vagus, with its branches (among which the depressor nerve, irritation of which would suspend the vasomotor tonus[3] over a

[1] We are still told that it is "the knowledge that the cervical sympathetic has a powerful influence upon the nutrition of the whole nervous system, which induces us to apply a pole to the ganglion cervicale superius." Now as physiology is absolutely silent on the subject (atrophy of the brain subsequent to section of the sympathetic being merely secondary effect of protracted disturbances in the circulation) this *knowledge* is either an assumption which we are free to accept or not; or at the best a hypothesis framed to explain the supposed clinical results of subaural galvanisation. In the latter case the argument is a simple *petitio principii*—a reasoning in a circle.

[2] Professor Vulpian's Classical Lectures on the "Système Vasomoteur" (1875), contain an exhaustive discussion, physiological and pathological, of the whole subject.

[3] Cyon ('Principes d'Electrothérapie,' p. 205,) who, as the discoverer of the depressor nerve, displays a very natural partiality towards it, tends to explain the assumed vasomotor effects of subaural galvanisation solely to an action upon this nerve. But it is obvious that currents strong enough to stimulate a nerve comparatively so distant, would act powerfully on the sympathetic as well.

The great Russian physiologist, ever ready to pour his withering scorn upon

very great area), and the base of the brain itself (the very structures, i.e. which are often supposed to be influenced indirectly through the sympathetic), receive their full share of the current. The importance of this diffusion of the current through various important structures, though occasionally recognised in principle in electrotherapeutical writings, is usually entirely lost sight of when we come to the clinical observations, and the indications for treatment. The effects observed on the heart's action—a diminution in the number of beats—are much more probably due to a stimulation of the vagus than of the sympathetic, whilst the tendency to sleep and giddiness are not ascribable to any, more than problematical, "vasomotor changes" ("anæmia or hyperæmia," believers are not quite sure which), but simply to some unknown, perhaps molecular, change in the brain-cells and fibres themselves. The sympathetic, like the vasomotor system, has truly become a refuge for the destitute of hypothesis, as Clifford Allbutt has happily remarked.[1] When it is objected to one of the adepts of the process that it is difficult to conceive how the stimulation of the cervical sympathetic can possibly effect the curative marvels we are bid to accept as due to its agency, it is triumphantly pointed out to us that the remarkable and extensive vasomotor and trophic functions of the nerve are quite enough to explain all, even "the absorption of adhesions into the medulla"! If, on the other hand, we object that we have no proof in experiments on the living man of the occurrence of such physiological effects as are readily observed in the laboratory, it is declared that of course it is wrong to expect almost homœopathic doses of galvanism administered to the nerve to have the same obvious results as an energetic

the pseudo-physiological sciolism of "les Electrothérapeutistes," indulges here in some pathological elucubrations very much in the style of those he abuses so roundly: "Most of these atrophies [progressive muscular atrophy, saturnine paralysis, &c.], arise from nutritive disturbances called forth by a spasm of the vascular muscles. These disturbances cannot be better arrested than by getting rid of the vascular spasm: the irritation of the depressor nerve, in paralysing all the vascular nerves of the body, must abolish any vascular spasm depending upon the sympathetic nerve." Irritation of the latter "could but increase the nutritive disturbance in the atrophied muscles, since it would necessarily augment the contraction of the vessels." . . . *Ipse bonus forsan dormitat Homerus!*

[1] 'Brit. and For. Med. Chir. Review,' 1871.

faradisation. We are left therefore to conclude that, probably owing to some form of neurility special to the superior cervical ganglion, it will react in a mysterious manner to the weakest currents, if only these be assisted by a vigorous belief in their efficacy. But we cannot do better than to conclude with a quotation of the author just named: "Here is a neck, and inside the neck is a sympathetic nerve-system; if we galvanise the neck, the current will flow somewhere about the tissues, according to their various conducting powers, and other physical conditions; therefore the sympathetic may probably come in for some of it, and we all know that a galvanised sympathetic causes contraction of the blood-vessels. Now there are blood-vessels all over the body, and in the brain, as elsewhere; so perhaps they will be kind enough to contract among others, and perhaps contracting [or dilating reflexly] may do the brain some good, though how, it is not clear. Meanwhile, if we see no change in the pupil, ear, or cheek, or but slight changes only, we must have faith and hope that more sensible changes are going on within. Surely this is the very midsummer madness of reasoning!"

THE LOCALISATION OF ATROPHIC PARALYSES.

BY DAVID FERRIER, M.D., F.R.S.

Motor paralysis may result from lesions of different parts of the neuro-muscular apparatus, each more or less distinctly characterised by special clinical features and concomitants. It may be a consequence of destructive lesion, temporary or permanent, of the cerebral motor centres, or motor tracts, or of the spinal centres and their centrifugal paths—the motor nerves, or of disease of the muscles directly. The forms of paralysis have been classified variously, e.g. cerebral, spinal, peripheral, and myopathic; or central, paralysis of conduction, and myopathic (Erb). For practical clinical purposes it is most convenient to differentiate paralyses according to the exact seat of lesion, whether in the brain, spinal cord, or peripheral nerves, &c.; but in reference to the question immediately under consideration we may divide paralyses of nervous origin into two great divisions, cerebral and spinal. Under cerebral we may include paralysis from lesion, either of the motor centres themselves, or of their conducting paths, whether in the internal capsule, crus, pons, or spinal cord. Under spinal we should include only paralysis from lesion of the anterior horns, or their conducting paths, viz. the peripheral motor nerves.

This division is made, because, altogether apart from the clinical features of each case, in the one group the paralysed muscles retain their normal physiological properties, and emaciate only from disease; in the other the muscles undergo degeneration as well as wasting.

In an ordinary case of hemiplegia depending on disease of the cortex or internal capsule, or the pyramidal strands, the

muscles, though paralysed as regards volitional activity, still retain their contractility and normal reactions to galvanism or faradism, and waste only from long-continued inactivity. If artificially stimulated, they may be maintained in an active state of nutrition for an indefinite period, subject to certain secondary changes due to spinal complications.

On the other hand, in cases of lesion of the anterior horns of the spinal cord, as in poliomyelitis anterior, infantile or adult, or in cases of lesion of the motor nerves of the muscles, not only are the muscles paralysed, but they speedily lose their normal electrical reactions, and undergo atrophic degeneration.

The question is, why do the paralysed muscles undergo degenerative changes in the one case and not in the other? and further, why do certain muscles and muscular groups specially suffer from atrophic paralysis?

The trophic degeneration of muscles which ensues on destruction of the anterior cornua or division of the motor nerves, is considered by many as an argument in favour of the existence of distinct trophic nerves and nerve-centres whose function is to preside over and regulate the nutrition of the tissues in which they are distributed. The facts are unquestionable, but the explanation is purely hypothetical.

The researches of Waller, confirmed by all subsequent observers, show that the motor nerves degenerate when separated from the spinal cord; and the sensory nerves degenerate if separated from the ganglion of the posterior root, the degeneration taking place on the side of section, peripheral or spinal. Muscles also undergo degenerative changes when separated from their nerve-centres, or when these are destroyed; but not only do the muscles degenerate under such conditions, but also bones and glandular tissues. Trophic disorders also ensue on lesions of purely sensory nerves. Ophthalmia from certain lesions of the fifth nerve, is well known, and herpetic and other cutaneous disorders, glossy skin, &c., are familiar in connection with injuries of nerves. But though neither the coincident vaso-motor disturbances alone, nor the loss of protective adaptivity consequent on anæsthesia, nor the propagation of ascending or descending neuritic processes alone, may suffice to give a satisfactory explanation of the

phenomena, we are not necessarily obliged to postulate the existence of a distinct system of nerves and nerve-centres called trophic.

Sir James Paget, in his highly philosophical lecture on Elemental Pathology,[1] has ably urged the necessity of recognising "the distinction between the conditions of life and the essential properties of living things," and the advantages to be gained by the study of the diseases of plants, organisms free from the complications of a nervous system. "It is not to be doubted that an organism such as ours, in which the work is more divided according to its kind, and more distributed to appropriate organs, is more subjected to regulation by central organs, and the working of each part is more influenced by that of all the rest; yet it is not probable that in any instance the law is abrogated according to which each elemental structure lives its own life in a method determined by its own inherent properties."

As in plants so in animal tissues, the higher the organisation the less the power of independent vitality and adaptation, and the greater the tendency to degeneration when they are left to themselves and cease to form related units of a complex organisation.

Sigmund Mayer[2] regards the central nerve-substance, the peripheral fibre and its peripheral end-organs, as contributing "not only a functional but a nutritive unity," or a mode of viewing the facts which seems to me preferable to the hypothesis of special trophic nerves and centres. We have as units of external function certain nerve-centres, centrifugal nerves and peripheral organs, muscular or glandular, and their adjuncts. In union they exhibit certain vital properties and reactions which we call normal. The muscles react in a certain definite manner to stimulation, mechanical and electrical. But dissolve the unity, and the tissues are left to their own powers of nutrition. The muscles degenerate and exhibit different reactions to stimulation; there are disorders of secretion, and the bones and connective tissues alter their state of growth. The more specialised the tissue, the greater the

[1] 'Brit. Med. Journal,' Oct. 16 and 23, 1881.

[2] Hermann's 'Physiologie,' Bd. ii. part 1; "Specielle Nervenphysiologie."

degeneration, while the more lowly organised tissues proliferate and luxuriate. The nutrition and growth of the nerve-tissue itself is dependent on the ganglionic cell of which it is merely the prolongation, and the nerve rapidly dies when the connection is severed. Functional inactivity, the parts all retaining their normal connections with each other, leads merely to defective, not to altered nutrition. The difference is one of degree merely, not of kind. Here there is simple atrophy, whereas in the former case there is, as Mayer terms it, allotrophy.

The units of internal function are certain peripheral organs adapted for the reception of different external agencies, and the centripetal tracts which connect them with the nerve-centres in which they abut and excite action. The nutrition and growth of the sensory nerves and tracts, and with them the normal nutrition of the peripheral organs and their annexes, are dependent on the ganglionic cells situated either at the extreme periphery, as in the retina, or at a varying distance, as in the Gasserian ganglion of the fifth, and the intervertebral ganglia of the spinal nerves.

Sever the connection with the ganglion cells, or destroy them, and all the related parts (peripheral and central) undergo degeneration. The laws of nutrition of the sensory organs and their annexes are changed, and various disorders consequent on altered vital conditions tend to manifest themselves. Especially is this the case under conditions of irritation. The sensory nerves and tracts degenerate up to the point of their primary functional manifestation. Some of these run only a short course in the posterior root zones, others ascend a long course along the columns of Goll, and others again ascend along the direct cerebellar tracts of Flechsig.

A similar system of functional and nutritive units obtains in the central nervous system. The cortical motor centres and the pyramidal strands abutting in the anterior cornua of the spinal cord form such a system. Destruction of the cortex, or severance of the path at any point between the cortex and the multipolar cells of the anterior cornua, leads to degeneration from the point of lesion downwards to its termination.

The anatomical differentiation of the functional units of the central sensory system still requires investigation, though these

also will no doubt soon be accurately determined in the hemispheres and encephalic ganglia.

We may from this point of view clearly represent the essential difference between cerebral and spiral paralysis. The functional and nutritive unit in the one case is the grey matter of the cortex, pyramidal strands and their terminations in the anterior cornua. The process of degeneration when the continuity is interrupted stops at the anterior cornua, which have an activity altogether independent of cerebral influence, though occasionally they also become involved in the degenerative process. The functional spinal unit is independent of the cerebral, and hence the muscles maintain their nutrition according to normal laws, notwithstanding the withdrawal of the influence of the cerebrum. But if the multipolar cells are destroyed, or the motor nerve cut, muscular degeneration is a necessary result.

Atrophy with degeneration or allotrophic wasting of the muscles may in all cases be attributed to lesion in some portion of the functional spinal unit. It may be in the multipolar cells of the anterior cornua, in the motor nerves, or in the muscular tissue primarily. Of the forms depending on disease of the cornua we have poliomyelitis anterior, infantile and adult. Of the second form, the phenomena of peripheral paralysis, traumatic, rheumatic, &c. The central origin of bulbar paralysis seems well established. To which form, central, peripheral, or myopathic, we are to refer progressive muscular atrophy and lead-palsy, are questions on which some important differences of opinion exist. Though the clinical features and general pathological anatomy of anterior poliomyelitis are well known, yet the special localisation of the lesion in cases where not a whole limb, but certain muscular groups only are affected by atrophic paralysis, is a subject on which at present very little precise information exists. It not unfrequently happens both in infantile paralysis and atrophic-spinal paralysis of adults, that only certain muscles are affected, while all other muscles remain in a normal condition; and similarly in the lower extremity. Apart from actual demonstration of the seat of the disease, the absence of sensory impairment, the escape of other muscles deriving

their nervous supply from the same trunk, and the coincident affection of muscles supplied by several different nerve-trunks, all exclude peripheral paralysis, and point either to affection of the anterior cornua, or some of the motor roots of the nerves supplying the limb.

The question then arises, whether certain portions of the cervical and lumbar enlargements, or whether certain roots of the brachial and crural plexus have definite and specific relations to certain muscular groups, or whether there is perhaps a complete differentiation in both senses.

Of the various researches which have been made on the lower animals with a view to determine the distribution of the roots of the plexuses of the limbs, those of Peyer and Krause, on the brachial plexus of the rabbit, are most deserving of attention.[1] These investigators found that most of the muscles of the limbs were supplied by more than one root of the plexus. Krause in his work, 'Anatomie des Kaninchens,' gives a tabular view of the various muscles related to each root. It is seen, among other things, that the muscles nearer the shoulder are supplied by the higher roots, and those of the hand by the lower roots of the brachial plexus, the 8th cervical and 1st dorsal.

The sensory roots have a corresponding superficial distribution over the regions of muscular distribution of the anterior roots.

These researches, taken in relation with others presently to be mentioned, have an important bearing on the question before us.

From a purely clinical and pathological standpoint, E. Remak[2] has recently advanced many able arguments in favour of the representation together in the grey matter of the spinal cord of those muscles which act in synergic combination.

Hence, for instance, the association of the supinator longus with the brachialis anticus and biceps, and the joint affection of these muscles along with the deltoid in what he calls his "upper-arm type" of atrophic spinal paralysis.

Besides the upper-arm type, he differentiates a "forearm

[1] For a more detailed account of previous researches on this question, see the communication by Dr. Yeo and myself in the 'Proceedings of the Royal Society,' No. 212; "On the Functional Relations of the Motor Roots of the Brachial and Lumbo-Sacral Plexuses."

[2] 'Archiv f. Psychiatrie,' 1876 and 1879.

type," which is essentially that seen in ordinary lead paralysis —paralysis of the extensors.

He has also attempted a differentiation of synergic muscular groups in the lower extremity; but the difficulties here are greater, both for want of material and in the way of exploration of the state of the individual muscles. But it appears that the sartorius frequently escapes, though the extensor cruris, supplied by the same nerve, is affected; and the tibialis anticus, according to him, not unfrequently goes with the extensor cruris.

With the object of throwing light on these topics by experimental research which might be considered as, with few exceptions, directly applicable to man, I have, in conjunction with my colleague Dr. Yeo, made a series of investigations as to the effects of irritation of the motor roots of the brachial and crural plexuses of the monkey. The results were embodied shortly in a paper read at the Royal Society.[1]

From these it is seen that each motor root represents a distinct functional combination; the muscles set in action being so correlated as to bring about a definite action clearly of an adapted nature. The relation is one of function, not of mere contiguity or community of peripheral nervous supply. Hence it is seen also that the purpose subserved by the plexiform junctions of the nerves of the limbs is to convey to the various muscles engaged in each functional synergy the requisite fibres related to each root.

The actions produced by the different roots may be briefly summarised as follows:—

Upper Extremity.

First dorsal;—action of the intrinsic muscles of the hand, muscles of ball of thumb, interossei, &c.

Eighth cervical;—closure of fist with pronation and ulnar flexion of wrist, retraction of the arm with extension of the forearm.

Seventh cervical;—the *scalptor ani* action, viz. adduction with rotation inwards and retraction of upper arm, extension of forearm and flexion of wrist and fingers so as to bring the tips against the flank.

[1] 'Proceedings of the Royal Society,' No. 212, p. 12.

Sixth cervical;—the movement of "attention!" viz. adduction and retraction of upper arm, extension of forearm, pronation and flexion of wrist, the palm of the hand being brought towards pubes.

Fifth cervical;—movement of the hand towards the mouth, viz. raising the upper arm inwards, flexion of the forearm with supination, and extension of the wrist and fingers.

Fourth cervical;—a similar movement of forearm and hand, but the upper arm is raised upwards and backwards.

The actions of the *lower extremity* are respectively:

Second sacral;—action of the intrinsic muscles of the foot, viz. adduction and flexion of the hallux, with flexion of the proximal phalanges and extension of the distal.

First sacral;—flexion of the leg, plantar flexion of the foot, flexion of all the toes at the proximal phalanges, and also of the distal phalanx of the hallux.

Fifth lumbar;—outward rotation of the thigh, flexion and inward rotation of the leg, plantar flexion of the foot and flexion of the distal phalanges.

Fourth lumbar;—extension of the thigh, extension of the leg and pointing of the great toe.

Third lumbar;—flexion of the thigh and extension of the leg.

The several muscles co-operating in these actions, so far as they could be observed and analysed, will be referred to subsequently.

Though the functional relations of the roots of the plexuses are indicated more or less precisely by our experiments, the question is not decided by them whether the motor nuclei of all the muscles engaged in each combination are in anatomical juxtaposition in the spinal segment corresponding to each root, in which case there would be several distinct nuclei for many of the muscles; or whether the motor nucleus of each muscle has a single definite position, not necessarily in the segment related to the root which calls it into action, but merely connected with the root by intracentral fibres running upwards or downwards as the case may be.

Thus the extensors of the wrist and fingers are specially related to the fifth and fourth cervical roots, but they are also related to the eighth cervical, and the question is whether the nuclei are only in the upper segments, or also in the segment corresponding to the eighth cervical.

These questions scarcely seem to admit of solution experimentally, owing to the complications which would necessarily be encountered, but the clinical and pathological facts relating to anterior poliomyelitis are more likely to throw light on the point. In the absence of actual demonstration *post-mortem* we cannot be absolutely certain that in a given clinical case of limited infantile or adult atrophic paralysis, the lesion is in the anterior cornu and not in a motor root of the plexus. We must rely on the ascertained facts and principles of diagnosis of anterior poliomyelitis in such cases. These seem to me rather in favour of the view that the motor nuclei of the several muscles excited by each root are anatomically related to each other in the corresponding spinal segment, so that there may be several spinal nuclei of the same muscle.

If this is the correct view, it will follow that a lesion of the grey matter of the anterior cornu accurately limited to a distinct segment will produce the same effect as lesion of the corresponding root. Hence those muscles will be most affected by atrophic paralysis which are innervated entirely or almost entirely by this segment; whereas those which are represented also in other segments will be affected to a less extent. The degree of atrophy will be in proportion to the degree of innervation.

A pathological lesion will rarely restrict itself accurately to the segment corresponding to each root, and more commonly we shall have affection of more than one. But theoretically we may differentiate the types of spinal monoplegia into as many as there are spinal segments and motor roots.

From an analysis of the muscles involved in each of the combinations already referred to, so far at least as we have been able to ascertain them as yet, we may indicate the following as the muscles which are likely to be affected in poliomyelitis strictly limited to each segment; the muscles being placed in the order in which they will probably suffer.

First dorsal type;—the intrinsic muscles of the hand, viz. muscles of the thenar and hypothenar eminences and interossei.

Eighth cervical type;—long flexors, ulnar flexors of wrist, intrinsic muscles of hand, extensors of wrist and phalanges, long head of triceps (pectoralis major?).

Seventh cervical type;—teres major, latissimus dorsi, subscapularis, pectoralis major, flexors of wrist and fingers (median) triceps.

Sixth cervical type;—latissimus dorsi, pectoralis major, serratus magnus, pronators, (flexor of wrist?) triceps.

Fifth cervical type;—deltoid (clavicular portion), biceps, brachialis anticus, serratus magnus, supinator longus, extensors of wrist and fingers.

Fourth cervical type;—deltoid, rhomboid, supra- and infra-spinatus (teres minor), biceps, brachialis anticus, supinator longus, extensors of wrist and fingers, diaphragm.

In the lower extremity we should have the following types:—

Second sacral type;—intrinsic muscles of the foot, strictly parallel to the first dorsal type.

First sacral type;—muscles of calf (plantar flexors), hamstrings, long flexor of big toe, intrinsic muscles of the foot.

Fifth lumbar type;—flexors and extensors of toes, tibial muscles, sural muscles, peroneal muscles, outward rotators of thigh, hamstrings.

Fourth lumbar type;—extensors of thigh, extensor cruris, peroneus longus, adductors.

Third lumbar type;—ilio-psoas, sartorius, adductors, extensor cruris.

No absolute accuracy is claimed for this order of enumeration, but as a general indication it may be accepted provisionally as a guide to further more minute clinical research. And it will be seen by the cases given below that clinical observation confirms in a substantial manner the distribution of the muscular affection in some at least of the types indicated.

It is quite possible for slight affection of some of the muscles forming a group, and also innervated by other roots, to escape detection by electrical exploration, and it will be necessary in future inquiries to test more minutely the relative strength of

the individual muscles, and compare them carefully with those of the other side.

As has been indicated, we can scarcely expect to meet with pure types; more commonly we are likely to have a blending of two or more contiguous ones.

The following cases which have recently come under my own observation are examples of an affection more common perhaps than any other spinal monoplegia of the upper extremity, viz. of the groups innervated by the upper cervical roots.

Case I.—Mary T., ætat. 45, a married woman, following the occupation of a sewing-machinist in connection with a tailoring establishment. This occupation involves holding and lifting heavy materials with the right arm.

A week before coming to the hospital—on November 13, 1880—she had had some pain in the right shoulder, but the movements of the arm were not affected. On Sunday November 12, on getting out of bed she found she could not raise her right arm.

On examination it was found that she was unable to abduct or raise the right arm away from the trunk beyond an angle of about 25°. Flexion of the forearm was very feeble. The arm could be flexed to a certain extent in the position midway between pronation and supination, by the aid of the muscles of the forearm attached to the condyles of the humerus. The shoulder could be elevated, but retraction upwards and backwards was very feeble on the right. Supination was much feebler on the right than left. The fingers could be clenched forcibly, and the wrist and fingers could also be extended. No comparison of the relative strength of the extensors was, however, made in this case.

The sensibility was in every respect normal.

The electrical reactions were tested on November 13, again on December 6, and on December 15. At the last date the condition was as follows:—

Circumference of upper arm.—Right, $9\frac{1}{8}$ inches; left, $9\frac{1}{2}$ inches.

Circumference of forearm an inch below condyles.—Right, $8\frac{3}{4}$ inches; left, 9 inches.

Acromio-axillary circumference.—Right, $14\frac{1}{2}$ inches; left,

15 inches, indicating wasting of the deltoid, flexors of forearm, and supinator longus.

The supra- and infra-spinous fossæ were also more distinct on the right side, and the muscles soft and flabby. Outward rotation of the right humerus impossible. Retraction and elevation of right scapula weak.

Faradic reactions.—Deltoid: excitability abolished in the scapular and acromial divisions, normal in clavicular portion. Excitability considerably diminished in the right biceps, brachialis anticus, supinator longus, rhomboid, and infra-spinatus: uncertainty as to the supra-spinatus, owing to the action of the trapezius.

No perceptible difference in the reaction of the serratus magnus of the two sides, nor of the extensors of the fingers.

Galvanic reactions.—Reaction of degeneration in the right deltoid (outer two-thirds), rhomboid, infra-spinatus and teres minor A C C > C C C. Excitability diminished in the biceps, brachialis anticus and supinator longus, but C C C > A C C.

Remarks.—This case diagnosed as one of limited anterior poliomyelitis corresponds almost in every detail to the fourth cervical type. The deltoid, the outward rotators of the humerus, the rhomboids, flexors of the forearm with supinator longus were distinctly affected, and in the proportion which one might expect, considering the degree of innervation of the respective muscles by the fourth cervical root. The extensors of the wrist and fingers were not perceptibly affected as regards electrical reactions, but their innervation by other roots, the fifth and eighth, would readily explain this.

Case II.—Daniel C., ætat. 65, a labourer, admitted at King's College Hospital in my out-patient department, on March 24, 1881.

The patient stated that a month ago on getting up one morning he found he could not raise his right arm. He had no pain in it. He had previously always enjoyed good health, and previous to his attack was not complaining specially.

The sensibility of the right arm was everywhere normal.

Abduction of the right arm from the trunk almost impossible. Cannot put his hand in the side pocket of his coat. Very feeble power of flexion of the right forearm. Extension

of the wrist and fingers of the right hand much feebler than on the left.

The electrical reactions on April 7 were as follows:—

Faradic.—Very feeble, almost abolished, reaction of right deltoid,—the clavicular and acromial portions being affected as much, if not more, than the scapular portion.

Excitability diminished in right rhomboid, infra-spinatus, serratus magnus, biceps, brachialis anticus, and supinator longus. Slight diminution also in the extensors of fingers and wrist.

Galvanic.—Excitability increased, but sluggish reaction, in the right deltoid, A C C > C C C.

Diminished in the right biceps, brachialis anticus, supinator longus, and extensors of the fingers, but C C C > A C C.

Remarks.—This case indicates an affection of muscles innervated both by the fourth and fifth cervical roots. The clavicular portion of the deltoid was here distinctly affected, as much if not more than the scapular. This portion seems specially in relation with the fifth root. The extensors of the fingers and wrist were also impaired.

The affection of the serratus magnus is also in agreement with what we should expect from the experimental data.

Case III.—Robert W., ætat. 17. Had worked as a plumber for a year. Came under my care at King's College Hospital on April 12, 1881. Nine months ago he began to have pain in his left shoulder and some difficulty in using his arm. This increased gradually up to the present condition.

Examined at this date, and again more thoroughly on April 28, and on June 13 the following condition presented itself:—

Considerable wasting of the left shoulder, upper and forearm as compared with the right.

Acromio-axillary circumference.—Left, 13½ inches; right, 13⅞ inches.

Middle of upper arm.—Left, 7¼ inches; right, 8 inches.

Forearm, 2 inches below olecranon.—Left, 7⅝ inches; right, 8¾ inches.

Complains of pain in the left shoulder, but the sensibility is normal. Flexion of left forearm very feeble, L. = 12 lbs., R. = 50 lbs. Diminished power of extension of left wrist also

as compared with right, L. = 12 lbs., R. = 18 lbs. Cannot rotate left arm outwards. The inferior angle of the left scapula is more prominent and stands 1½ inch higher than that of the right.

Electrical reactions: Faradic.—Deltoid, much diminished generally. The clavicular portion reacts more readily than the rest.

Diminished also in infra-spinatus, serratus magnus, biceps, brachialis anticus, supinator longus, rhomboid, pectoralis major and latissimus dorsi.

The excitability was increased in the extensors of wrist and fingers.

Galvanic.—Deltoid: greatly diminished in all parts. The clavicular portion reacted more readily. No qualitative change.

Diminished reaction also in the infra-spinatus, biceps, supinator longus and the other muscles above mentioned. No qualitative change. Excitability of the extensors of wrist and fingers increased, but C C C > A C C.

Remarks.—This case is also a combination more or less complete of the muscular groups innervated by the fourth and fifth cervical, and also by the sixth. Nine months having elapsed since the onset, it is difficult to determine what the conditions of the respective muscles were in the early stages. But there were abnormalities, as above indicated, present in those muscles which we have seen act together on stimulation of the fourth and fifth and sixth cervical roots. In this case there was some suspicion of lead-poisoning. The patient had not, however, worked long as a plumber, he had never had lead colic, yet there was a suspicious-looking line at the margin of the gums. He had not had drop-wrist. The fact, however, is important, and will be referred to subsequently in connection with saturnine paralysis.

Case IV.—Sydney W., ætat. 15. Admitted as out-patient at the National Hospital for Paralysed and Epileptic on April 8, 1881.

On January 1880 complained for several days of pain in the left shoulder. In the summer the arm became weak, and has been more or less paralysed ever since.

Cannot raise the left arm from trunk, nor draw the left shoulder upwards and backwards. Flexion of left forearm very feeble. Cannot place his left hand on right shoulder; cannot cross his wrists behind his back.

There is wasting of left shoulder, hollowing of the supra- and infra-spinous fossæ, thinning and flabbiness of biceps and supinator longus. The lower angle of left scapula is more prominent than the right when the arms are hanging down by the sides. This becomes much more pronounced when the arm is raised.

The sensibility was normal.

Faradic reactions —Deltoid, sub-normal in clavicular position, almost abolished in acromial and clavicular portions. No action obtainable in left infra-spinatus, very doubtful action in left rhomboid. The right contracted vigorously to same current. Diminished action in left biceps, supinator longus, serratus magnus, pect. major, latissimus dorsi. Slight diminution in the extensors of fingers.

Remarks.—This case is almost an exact repetition of Case III. —the upper cervical type.

These cases show that in addition to the usually recognised combination of affection of the deltoid, flexors of forearm and supinator longus, constituting Remak's upper arm type, other muscles also suffer more or less in accordance with the grouping indicated by experimental research. The outward rotators of the humerus and rhomboids go with the deltoid, more particularly when its outer two-thirds are affected, and the serratus magnus, which is a synergic muscle with the deltoid, likewise suffers, especially when there are indications of affection also of the clavicular portion of the deltoid. The escape of the clavicular portion of the deltoid, which is so commonly noted even when the scapular and acromial portions are affected, receives its explanation in the fact that it goes more with the fifth than with the fourth cervical root.

The extensors of the wrist and basal phalanges are also shown to be liable to be affected in combination with the supinator longus, flexors of the forearm and deltoid, though to a less extent relatively than these, and more particularly when the affection implicates both the fourth and fifth more or less.

The comparatively slight affection of the extensors even in this case seems to be satisfactorily accounted for by the representation of the extensors again in the eighth cervical root, as synergic with the flexors of the fingers. This fact would, if this is the correct view, support the notion that the motor nuclei of the extensors are not anatomically restricted to a single definite position in the anterior cornua of the cervical enlargement, but are represented in each segment corresponding to the motor root which calls them into action synergically with other muscular groups.

Otherwise it would be difficult to account for the relatively slight affection of the extensors of the fingers, when the other muscles of the groups are affected to a very marked extent, either on the theory of direct affection of the fourth and fifth motor roots, or on the more probable myelitic nature of the lesion in the above-recorded cases.

The above localisation affords an explanation also of the peculiar "main à griffe," described by Charcot and Joffroy in connection with hypertrophic cervical pachymeningitis. Here we have a very marked extension of the wrist and basal phalanges with paralysis and atrophy of the muscles supplied by the ulnar and radial nerves. This will occur when the pachy-meningeal lesion is limited more especially to the lower portion of the cervical enlargement, leaving the upper segments specially related to the extensors free, and hence the unantagonised action of these muscles and the characteristic "griffe," with hyperextension of the wrist.[1]

On the other hand, when the pachymeningitis affects the upper half of the cervical enlargement, as in a case reported by Dr. Leech, and figured by Dr. Ross,[2] we have exactly the opposite condition, viz. unantagonised action of the flexors and pronators, the muscles supplied by the musculo-spiral being specially paralysed.

[1] See figure in Charcot's 'Leçons.' Troisième Partie, 1874, p. 251.
[2] 'Diseases of the Nervous System,' vol. ii. p. 388.

(*To be continued.*)

Critical Digests and Notices of Books.

Dr. Leonardo Bianchi *on the Significance of Electric Excitation of the Cortical Motor Zone.*

From thirty-seven experiments the author comes to the following conclusions:—

1. That there exist in the cerebral cortex cellular excito-motor elements capable of being experimentally excited by electricity.

2. That these elements, if indeed they are to be found disseminated through the whole cortical grey substance, are yet grouped in such a manner in the cortical motor zone as to constitute of themselves alone true cortical centres, and the only ones which experiment has succeeded in discovering.

3. These centres are true psychic motor centres, distinct from the centres for reflex cerebral movements, and also from those for automatic movements.

4. It is in no way proved that the functions performed by the voluntary motor centres can be carried on by any other cerebral organs. On the contrary, this seems highly improbable.

5. It is equally a question whether these centres are composed entirely of motor elements, which receive impulses from other elements in which volitional determination and the idea of movement are produced, or whether these powers reside in them conjointly with the faculty of developing excito-motor force.

6. However this may be, the bundle of fibres proceeding from them pass exclusively into the internal capsule (its two anterior thirds); and a lesion of the latter produces a hemiplegia probably quite identical even in animals with that produced by destruction of the motor cortical zone.

7. It appears to be proved that the cerebellum is not directly concerned in the course followed by the electric stimulus applied to the motor zone, in order to produce the phenomena of which we have been treating.

The author, having already in 1878 reviewed the history of the experimental demonstration of the cortical motor centres, does not re-discuss this part of the subject. Neither does he consider the question of the production of movements through centres for organs of sense, confining himself to movements of the head, face, tongue, mouth, and limbs. The corresponding centres are located about the fissure of Rolando in man and apes; about the cruciate sulcus in dogs, cats, and sheep (though the sulcus is less marked in the last) in corresponding parts, although apparently in different localities in the solipeds; and in parts approximately correspondent in the rabbit, whose brain shows only a trace of the cruciate sulcus.

The first experiments were made with the view of testing the opinion that movements obtained on stimulating the cortical motor zone were due to irritation of the ganglia at the base of the brain. If this were so, the more nearly the stimulus approached these ganglia the more marked should be the movement. In experiments 1, 2, 3, the brains of two dogs and a rabbit were pierced by electrodes, convolution by convolution, and point by point, on the lateral parts of the hemisphere, and also (in the dogs) upon the internal aspect of the same, on the point nearest to the corpus striatum, but without the slightest effect being produced in any of the experiments. It was not possible to cause isolated movements of one limb or of distinct groups of muscles by irritating the corpus striatum directly.

Experiments 4, 5, 6, 7. On penetrating with fine platinum needles, covered as far as the point with a layer of vulcanite, to the caudate nucleus and, in a different direction, to the corpus striatum, there followed, not the definite movements of special parts obtained on stimulating the motor zone, but this: a sudden tonic contraction of all the muscles of the opposite side. The thigh flexed on abdomen with the limb and foot extended, the anterior limb carried forward, rigid; the trunk laterally curved as in pleurosthotonus, the head

turned to the opposite side. These movements differed in character from those got by stimulating the cortical motor zone, being more rapid or instantaneous, and not being apparently adapted to any determined end.

Experiments 8, 9, 10 and 11 show that the course taken by a stimulus commencing in the cortical motor centres is through the internal capsule. When this is divided, no movements follow, even although the basal ganglia and all their connections with the cerebral cortex remain intact. In one of the experiments, section of the capsule was not quite complete, and then movements in the anterior extremity followed stimulation of the cortical zone. These consequences of division of the internal capsule agree with the anatomy of the part, and in particular with the existence of the large pyramidal cells found in it by Betz and Mierzejewski.

Experiments 12 and 13 show that stimulation just in front of and just behind the motor zone is not followed by movements under moderate stimulation, although stimulation by a strong current may induce epileptiform convulsions.

Experiment 14 is very important. Dr. Bianchi chloroformed a dog, laid bare the motor zone, and obtained the usual movements. The animal meanwhile came out of the chloroform. On giving it more chloroform, the respiration suddenly ceased. He reapplied the current more strongly than at first, and obtained once only the same circumscribed movements. In the meantime, on attempting to revive the animal he found it dead.

The author says that this experiment proves that the animal could not have have had any sensation by which it retracted its limbs. It is known, he says, that in the deepest anæsthesia from chloroform or ether the electric contractility is little changed, and endures for some time even after death. All the nervous mechanism of sensation is paralysed long before the apparatus of motion, "and my observations," he says, "whose significance I cannot overlook, although they are opposed to those of Schiff, confirm definitely the fact that the electric irritability of the motor nervous apparatus resists narcotic influences much longer than the sensory apparatus."

In reference to this important statement, it may be observed that in giving patients chloroform for surgical operations or in

obstetrics, they continue to move, to flinch from the knife or the hand, and also to talk, laugh, sing or pray, long after they cease to know they are doing these things. At least, when questioned after their return to consciousness, they say they remember nothing of them. These facts seem to corroborate Dr. Bianchi's views. But, on the other hand, it should not be overlooked that another explanation is possible; this namely. It may be the case, as Dr. Munk suggests, that the *perceiving* centres are not the *remembering* centres, and therefore persons who do not remember saying or doing certain things may yet have perceived them when they did them. Dr. Bianchi's dog, in short, may have *perceived* or *had the sensation of* the electric stimulus by its perceptive centres, even after the *remembering* or *judging* centres were dead.

In experiment 18 he removed the grey substance of the cortical motor zone, and then by stimulation he obtained the usual movements, only more rapid and rather tending to be tetaniform. From this he infers that no sensation of contact can have preceded the movement of the limb, since the apparatus of sensation was removed. This is of course the case, but still it scarcely follows that there is no sensation when the apparatus is perfect, as in ordinary circumstances. All that he is warranted in inferring seems to be that the movements can be produced by stimulating lower centres than those of the cerebral cortex, a fact which, of course, is not new. But then the movements tend to become tetaniform. That is, on the opponent's supposition, just because the controlling sensation of contact is absent.

Dr. Bianchi says that tactile sensibility is not diminished by removal of the cortical zone. This may be, and yet it may be true that perception of that tactility may be not only diminished, but destroyed by that removal.

In experiment 19 the author destroyed the cortical motor zone in a rabbit, after having determined by the electric current the centres for the movements of retraction of the angle of the mouth. After removal it was found that the movements were no longer obtained. After that, he passed the pulps of the fingers over the ends of the hairs fixed around the mouth, and the rabbit frequently turned his head from the

opposite side, so affording the most indisputable evidence that the tactile sensibility was intact.

This experiment, the author says, is totally subversive of the reflex theory; but, supposing the *whole* centre to have been removed, would not passing the fingers *around* the mouth (intorno al muso) excite other reflex functions, besides the functions of those removed? And lastly, even if centres cannot act when removed, does it follow that they do not act in normal circumstances when the mechanism is perfect?

Experiments 20, 21, and 22 relate to the action of strychnine and lead to a discussion of the four propositions laid down by Brown-Sequard. 1. The symptoms in organic affections of the brain do not arise through the loss of a function belonging exclusively to the part destroyed, nor through the direct effect of a manifestation of the special property of that part. 2. On the contrary, these symptoms are only the consequence of an influence exercised upon other parts at a distance greater or less from the seat of the organic lesion visible at the autopsy, an influence caused by an irritation transmitted either by the very part destroyed or by the neighbouring parts. 3. Paralyses, amauroses, anæsthesias, aphasia, are produced by a mechanism identical with that by which the heart is arrested by irritation of the vagus. 4. The mechanism of the manifestations of exaggerated activity, delirium, epileptic and choreic convulsions, vomiting, &c., is the same as that by which all these phenomena are produced, since their original cause is a peripheral irritation to the skin, to the mucous surfaces, or to some point in the course of a sensitive nerve.

The author does not care to follow Brown-Sequard into the wide domain he occupies, but confines himself simply to the consideration whether there exists a motor zone in the cerebral cortex. This being so, the only question for consideration is whether the symptoms consequent on irritation or on lesion of the cortical motor zone depend on an influence exercised upon other parts at a distance from the seat of the irritation or visible lesion. The action at a distance may be conveyed either to other parts of the cerebral cortex, or to the ganglia and other parts at the base. But as in men and the higher animals the basal ganglia govern only automatic movements,

the action by other parts of the cortex is the only one to be considered.

In order to determine this question, experiments 23, 24, 25 and 26 were made. In these experiments a given centre, say that for the anterior limbs, was isolated from the rest by a platinum plate which could be heated by a galvano-caustic apparatus. This being plunged into the brain as far as the white substance and then heated to redness, effectually separated the centre from the others. But on reapplying the current, the accustomed movements occurred.

In experiments 27, 28, and 29 the same separation was effected by the knife, the animal being under the influence of chloral, but still the ordinary movements occurred.

Experiments 36 and 37 relate to the action of the cerebellum, and seem to the author to justify the conclusion already detailed as No. 7.

The remarks on Dr. Brown-Sequard's views are exceedingly interesting, but space forbids our entering on them. If the author has not solved the difficult questions connected with the discussion of cerebral functions, his experiments are well conceived and carefully executed, and may, on the whole, be said to have advanced our knowledge. A. Rabagliati.

Betz *on the Structure of the Cortex Cerebri.*

In an article contributed to the 'Centralblatt' for August 1874,[1] Professor Betz has given the details of an extensive examination of the cerebral cortex, which proved a valuable addition to our knowledge of its histology, as he therein advanced several original observations indicating the existence of certain definite regions hitherto unnoticed amongst the list of those characterised by a dissimilar lamination. With a few exceptions and misstatements, this article was a really fresh addition to existing knowledge of cerebral topography. In the March number of the same journal for the present year[2]

[1] "Gehirncentra." 'Centralblatt f. d. Medicin. Wissensch.' Nos. 37 and 38, 1874.

[2] "Feinere Structur der Gehirnrinde des Menschen." 'Centralblatt,' Nos. 11, 12, 13. 1881.

there appears an account of a still more extended inquiry into the diversified structure of the human cortex cerebri, which contains so much in which Professor Betz has been wholly anticipated by the labours of other inquirers without any recognition of such work, that we naturally wonder at the author's neglect in this respect. It is therefore thought advisable, in a critical survey of his two articles, to review the facts which have been advanced by others, and practically ignored by Professor Betz. In his earlier memoir the following conclusions are arrived at.

1. The whole cerebral cortex is divisible into a motor and sensory portion approximately divided by the fissure of Rolando.

2. The motor division is characterised by the prevalence of large pyramidal cells; the sensory by the prevalence of so-called granule cells.

3. A constant lobule exists in man and the higher apes upon the median aspect of the hemisphere at the extremity of the central gyri, which he names the paracentral lobule.

4. This lobule is characterised by the presence in its cortex of the largest nerve-cells of the brain, hence termed "giant pyramids."

5. These cells closely resemble the motor cells of the anterior cornua of the spinal cord.

6. Such cells are found in a similar position in man, chimpanzee, ape, and baboon.

7. They likewise exist in the gyrus surrounding the crucial sulcus in the dog. (Sigmoid gyrus.)

8. The size of this lobule bears a peculiar vicarious relationship to the development of the central gyri; the one being large and complex when the other is but poorly developed, and *vice versâ*.

9. This sigmoid gyrus (found electrically excitable by Fritsch and Hitzig) is not the analogue of the central gyri of man and apes, but of their paracentral lobule.

After a short account of the sensory division of the brain, which is only too meagre in detail, our author sketches lightly the resemblance betwixt the rough conformation of the cerebral hemispheres and the spinal cord as regards its bilateral,

motor, and sensory segments, its commissural connections, &c. Referring to the peculiar site of the "giant cells," which he regards as motor, and their relationships to the great venous sinus, and large veins, he suggests this as a plausible explanation of many cases of transitory paralysis of centric origin, of puerperal eclampsia, epileptic spasm, hysterical laughter, partial and complete aphasia.

Prior to the appearance of this memoir by Betz, these great cells had been noted by several observers, and were regarded as "hypertrophied nerve cells;" but to Betz is due the credit of indicating their constant presence in healthy brain, and their definite localisation in the paracentral lobule. In claiming for them a motor significance, however, he simply followed out the idea expressed years previously by Meynert,[1] that the cells of the third layer closely resemble those of the anterior cornua of the cord, and the nuclei of the motor cerebral nerves. The points to which objection must be taken in the above article refer to—

a. The distinctive appellation of "giant cell."
b. The position of these cells in the cortical layers.
c. Their distribution over the superficies.
d. Statements regarding the gyri of carnivora.

I have elsewhere[2] stated my conviction, together with that of a fellow-worker, that the term "giant pyramid" is inapplicable to these cells, since they were but great representatives of a very general formation varying greatly in size. In the article referred to we stated that "the disparity in size between these elements in the fourth layer of the motor area induces us to reject the term 'giant cell' as applied *generally* to this formation, and to call them by preference the ganglionic cells of the cortex."[3] It was shown by us that these ganglionic cells were not only extremely varied in contour, and not by any means constantly pyramidal, but that they were represented over a much greater area of the brain than was affirmed by Betz—in fact, were spread over the whole vault where in

[1] 'The Brain of Mammals,' by Th. Meynert. Stricker's 'Human and Comp. Histology,' Sydenham Soc. vol. ii. p. 387.

[2] "Cortical Lamination of the Motor Area of the Brain." Proceedings of the Royal Society, No. 185, 1878.

[3] Op. cit.

most positions their dimensions did not exceed the lower cells of the third layer. From these facts it will be seen that the term used by Betz is inadmissible.

Again, as regards their position in depth, it was shown that these peculiar cells could not be regarded as invariably situated in the *fourth cortical layer*, as over the greater area of the vault the cortex was distinctly *six-laminated*, and these cells consequently would here occupy the *fifth layer*. It was shown that Betz[1] and Mierzejewski,[2] following Meynert,[3] were wrong in regarding the general type of cortical lamination as five-layered, whilst Baillarger was correct in representing it as six-laminated. The true position is, as we first indicated, that there was both a five and a six-laminated cortex, the former specially characteristic of Ferrier's motor area, the latter of sensory realms where the interposition of a granule belt formed a six-laminated cortex.

CORTICAL LAMINATION OF VAULT.

	After Meynert, Betz, and Mierzejewski.	Baillarger.	Lewis and Clarke.	
			Motor Sensory.	Motor Sensory.
1st Layer .	The Neuroglia zone.	Do.	Do.	Do.
2nd „	„ Small pyramidal cells.	Do.	Do.	Do.
3rd „	„ Large pyramidal cells.	Do.	Do.	Do.
4th „	„ Irregular granule cells.	Do.	Ganglionic cells.	Granules.
5th „	„ Spindle cells.	Large pyramids.	Spindle cells.	Ganglionic.
6th „		Spindle cells		Spindles.

The position held by us is one of importance, since by it the sensory realms are shown to acquire a new layer by the intercalation of a belt of granule cells *above* the ganglionic formation. Later on, in a memoir published in 'BRAIN,' it was shown that the belt of granule cells was richly developed in proportion to the fading off of the ganglionic series, and *vice versâ*. Whilst fully admitting their nested arrangement and other special features described by Betz, we wholly dissented

[1] Op. cit.

[2] 'Archives de Physiologie,' 1875. "Études sur les lésions cérébrales dans la paralysie générale."

[3] Op. cit.

from their limitation to the narrow districts which he assigned them. Exception must also be taken to his statements concerning the analogue of the central gyri in carnivora. Professor Betz concludes, from a consideration of the minute structure and relationships of the paracentral lobule in man and the higher apes that its analogue is to be found in the sigmoid gyrus, and that the latter does not represent the central gyri of higher animals and man—"the central gyri of dogs being wholly absent." This latter assertion is quite unwarranted, since, by a far more philosophical method of inquiry, Broca has shown that the central gyri, separated by a well-marked sulcus (Rolando),[1] are reproduced at the frontal pole of the brain in carnivora. According to Betz, the frontal end anterior to the "motor region" is the representative of the frontal gyri, and no true Sylvian fossa is shown. The former statement is in every way inaccurate; and as to the latter, a Sylvian depression is certainly seen.

In his later memoir our author states that hitherto neither the researches of Fritsch and Hitzig nor those of Ferrier have been afforded a basis in the anatomical construction of the brain, and advances the results of his recent investigations as supplying the deficiency; thus totally ignoring the classical works of Charcot and of Meynert, which most strongly point in this direction; as also my own investigations on the cortex cerebri, published between the years 1878 and 1880. Preparatory to giving the details of his work he describes the general laminar type so as more clearly to establish the divergencies which occur. He again reiterates his earlier statement that this type is *five*-laminated, but his description or summary is both defective in detail and exactitude, and cannot be fairly contrasted with the descriptions afforded by other authorities, Meynert, &c. &c. Thus in alluding to the first layer he speaks of it as simply an extension of neuroglia, totally ignoring its complicated nervous network derived from the subjacent nerve-cells, as well as its peripheral medullated zone and corpuscular elements. Then follows a summary of the varieties of cortical structure, based upon the examination of 5000 preparations

[1] "Anatomie Comparée des Circonvolutions," par Paul Broca. 'Revue d'Anthropologie,' 1878.

from the right and left hemispheres of the same individual; also from the brain of male and female, young, adult and aged subjects; as well as in the brains of a seven months' fœtus, a new-born child and an infant six weeks old.

The whole cortex cerebri, which he previously divided into a motor and sensory portion, is now subdivided by him into the following realms, differing from one another in histological constitution :—

1. Ascending frontal gyrus.
2. Gyrus fornicatus.
3. Cornu ammonis.
4. Third or inferior frontal gyrus.
5. Paracentral lobule.
6. Lingual gyrus.
7. Tip of the temporo-sphenoidal lobe.

Let us contrast with this enumeration Meynert's five types of cortical lamination, which are as follows :—[1]

1. Type common to vault and gyrus fornicatus.
2. Occipital apex.
3. Sylvian fissure.
4. Cornu ammonis.
5. Bulbus olfactorius.

We here see that Betz has subdivided Meynert's first district into three subdivisions, whilst his lingual gyrus really embraces the same type of structure as Meynert's occipital type. The object of Meynert's enumeration was to present us with generic divergencies in structure, and this he has faithfully done; for all his types, with one exception, allude to cortex which differs in kind rather than in degree, in the actual absence of certain layers or addition of new layers. Betz, however, appears to dwell chiefly upon the more minute specific differences in the layers and their elements, and with this object has dwelt upon each individual convolution; yet in his summary, as given above, he has inextricably confused the fundamental distinction to be always kept in mind, between areas whose structures vary in kind or in degree; and thus such wholly different structures as the cornu ammonis, lingual gyrus, tip of temporal pole, are

[1] Op. cit. p. 381 (published 1872).

mixed up with the various regions which he regards as five-laminated. We would insist strongly upon the necessity of upholding the above distinction, otherwise results given by individual inquirers must be confusing and conflicting. Nor do we see why he should exclude (upon his own principle) the olfactory bulb from his tabulation, as it is as surely a portion of the cerebral cortex as the cornu. Space forbids a detailed account of each individual gyrus as given by Betz, but as regards the site of the "giant pyramids," it is now stated that typical nests of these great cells are found in the paracentral lobule, and extend over the upper end of the ascending frontal gyrus—deep in the fissure of Rolando at its median extremity and very exceptionally on the anterior surface of the ascending parietal. It is here stated that giant cells of "small calibre" are found grouped at the origin of the two upper frontal gyri—also at the posterior end of the lowest frontal, extending back into the ascending frontal. This localisation by Professor Betz confirms in a remarkable way the results published by the reviewer two years ago.[1] A critical survey of the divergencies in structure of the cortex in the various gyri unfortunately affords us very little additional information. The peculiarities in structure of the lingual gyrus, of the temporo-sphenoidal lobe, and of the olfactory bulb were all known and long since described by Meynert, and the more trivial differences in most of the other gyri (with exception of the more special motor realms) simply obey the law expressed by the writer some years since, as follows: "No *abrupt* passage from one form of cortical lamination to that of another is ever seen in the convolutions of the vault. Changes in type are gradually assumed, and no distinct boundary exists to indicate an exact line of demarcation. The structure peculiar to one region gradually fades and blends with arrangements which foreshadow the architecture of adjacent realms, and thus we obtain *transition regions*."[2] It is in the enumeration of such transition regions that Professor Betz chiefly engages when

[1] Loc. cit.

[2] "Comparative Structure of the Cortex Cerebri;" Bevan Lewis, in 'BRAIN,' Part I. 1878, p. 84. 'Proc. Roy. Soc.' No. 185, 1878; and in 'BRAIN,' Part I. p. 83.

not describing the really typical varieties of the cortex. Again, in his very cursory examination of the structure of the insula, the features of this important region are given in a most vague and inexact manner, nor is any reference made to the very thorough examination of its lamination which was described by Dr. Major in 1876.[1] It is much to be regretted that in this later memoir the same scientific exactness does not appear which characterised the former article and which was its especial charm. Our author had apparently entirely discarded the use of the micrometer, and the vague expressions, larger, smaller, thick, or thinner, are constantly employed; and his results are therefore to a great extent removed from the test of comparison with those of other inquirers. This is all the more to be regretted, as the special portion of his work which is stamped by greater exactness, viz. that of the localisation of the nested "giant cells," is so substantially a confirmation of what had previously been advanced. There are other points of importance upon which we must join issue with our author. In his former memoir he describes the "giant cells" as specially localised in the paracentral lobule and upper end of the ascending frontal; in the later memoir he extends their limits, and refers to these distant elements as "giant cells of small calibre,"—surely a most inappropriate term when these elements resemble in size the cells of the lowest stratum of the third layer. We are safer in terming them ganglionic cells, and in recognising their existence over the greater portion of the vault, and especially decreasing in size towards the lower end of the ascending frontal (*vide* Measurements).[2] Again, we must strongly insist upon the point that the *general type* of cortical lamination is of six, and not five layers; and would suggest as the cause of conflicting opinion on this point the neglect of fresh examinations of the cortex.

The method adopted of distinguishing the individual layers by number is another element of fallacy; and far less confusion would be introduced if each layer was named after its prominent morphological character. Thus I am in the habit

[1] 'Histology of the Island of Reil.' Dr. Major, 'West Riding Reports, 1876.'

[2] "Comparative Structure of the Cortex Cerebri;" Bevan Lewis, in 'BRAIN,' Part I. 1878, p. 84. 'Proc. Roy. Soc.' No. 185, 1878; and in 'BRAIN,' Part I. p. 83.

of describing the *general cortical type* as consisting of the following layers:—

1. Peripheral cortical zone.
2. Small pyramidal layer.
3. Large pyramidal layer.
4. Small angular or granule layer.
5. Ganglionic layer.
6. Spindle layer.

W. Bevan Lewis.

Bernhardt on Cerebral Tumours. (Beiträge zur Symptomatologie und Diagnostik der Hirngeschwülste.) Von Dr. M. Bernhardt. Berlin, 1881. 8°. pp. 336.

This work is a laborious abstract and tabulation of the symptoms which have been recorded by different observers in connection with tumours situated in various parts of the encephalon. The list is extensive, if not complete, and though the cases are not reported with such a degree of fulness as to dispense with the necessity of referring to the originals, yet clinical workers will be thankful to Dr. Bernhardt for saving them from much tedious labour. Beyond the valuable collection of facts, there is little calling for special remark. Bernhardt's work has been anticipated, and we think greatly surpassed in lucid arrangement and treatment, in the chapters devoted to cerebral tumours in Nothnagel's treatise on the Regional Diagnosis of Cerebral Disease.

D. Ferrier.

Clinical Cases.

CASE OF BRAIN LESION WITH HEMIPLEGIA ON THE SAME SIDE AND UNI-OCULAR NEURITIS ON THE OPPOSITE SIDE.

BY ERNEST FIELD, M.D., BATH.

H. R., æt. 26, first came under my care in June 1879, with the following history:—Five or six years before he contracted syphilis, but with that exception had enjoyed good health until June 1876, when he had a slight attack of hemiplegia on the right side. This occurred during the night, and was only noticed by him on waking in the morning. It was, however, not enough to confine him to bed beyond a week, and at the expiration of that time he was able to walk, with assistance, to the hospital, where he remained as an in-patient for a week or two.

He recovered sufficiently to return to his work, at which he continued more or less up to May 1879, though his intellect became much duller, and he suffered throughout from some weakness of the limbs of the right side.

His condition when he came under my care, was as follows:—Distinct, though slight, loss of power in the right arm, with a little dragging of the corresponding leg; no loss of sensation on this side, but in the left thigh sensation distinctly diminished, and in it only; some drooping of the left eyelid, with complaint of occasional diplopia.

On both legs were the scars of old rupia.

He lay in bed in a peculiarly lethargic, somnolent, condition, with scarce sufficient power to raise himself, and from time to time had retention of urine, for which the catheter was necessary; fæces occasionally passed involuntarily, but only after the administration of purgative medicine, which the obstinate constipation from which he suffered rendered necessary.

Eyes normal, beyond irregular dilatation of pupils on introduction of atropine, the result of part iritis.

He was ordered perchloride of mercury, iodide of potassium,

and tonics, and under this treatment he slowly improved and was again able to get about; but during the next twelve months he was subject to these attacks of lethargy and general loss of nerve power, removed by a return to specific remedies.

For the four months preceding his death he was able to walk down to the dispensary, a distance of half a mile, and he did this up to two days before his fatal seizure. During this last period he had an attack of optic neuritis, confined to the left eye, with hæmorrhages on and around the disc, but from this he completely recovered, with good vision.

An examination of the knee-reflex about two months before death showed it to be present in an exaggerated form in the right leg, but absent in the left. No clonus at the ankle was obtainable on either side, but there was great resistance to dorsal flexion of the foot.

Epileptiform attacks were present during the last year of life, but I was never able to see him in one. On the morning of his death he was found in bed in a comatose state, breathing stertorous and irregular, pupils dilated but equal, pulse 140; and after a few hours he died.

The diminution in sensation noticed in the left thigh had quite disappeared for some months before death, and the only paralysis existing was the slight affection of the right side before described.

Pain in the head was not a prominent feature at any time, though on being questioned as to whether he had any, he usually replied in the affirmative.

At the *post-mortem* examination I had the kind assistance of my friend Dr. Berry, at that time house-surgeon to the Eastern Dispensary.

On removing the calvarium the contents seemed tightly packed and the vessels somewhat engorged. In the ventricles and spinal canal was a large quantity of cerebro-spinal fluid. At one spot on the right side, in the situation of the operculum, the dura mater was firmly adherent to a hard substance at the surface, occupying an area about the size of a half-crown piece; but the whole hemisphere around this was much softened—in fact, quite diffluent, only the occipital lobe and the extreme anterior part of the frontal lobe were normal in consistence. This extreme and widely-spread softening rendered exact localisation of the tumour somewhat difficult. The growth was of a slaty appearance on section, and reached the surface in the region of the operculum; of irregular, nodulated shape, it was in all about the size of a pigeon's egg.

The left hemisphere was fairly normal. In the ependyma covering the corpus striatum was a small, yellowish thickening at one spot, the area of a shirt-button. Neither corpus nor

thalamus was quite as plump and large as usual, but there was nothing very marked wrong with them.

On microscopic examination the tumour was found to be composed of small round cells, with a delicate reticulum, resembling imperfectly developed adenoid tissue. In parts much caseous change had occurred.

Remarks.—There are two features of special interest in this case, viz. the fact that the hemiplegia and the brain lesion were on the same side, and the occurrence of uni-ocular optic neuritis.

As is well known, Flechsig has pointed out that there is great variation in the amount of decussation which takes place in the anterior pyramids ; whereas usually the larger portion of fibres cross over to the opposite side of the cord, passing down in the posterior part of the lateral column, whilst the smaller portion only remains on the same side, constituting the anterior median column, yet occasionally this is not the case, and the mass of them remain on the same side. Whether any such arrangement obtained in the present case I am unable to say, but without it there is much difficulty in explaining the phenomena observed.

The usual difficulties attending autopsies in private houses deterred me from removing the cord; but it is much to be regretted that this was not done, as the increased knee-reflex on the affected side rendered the existence of some descending degeneration probable, whilst the position of the latter might have thrown some light on the decussation which had occurred in the anterior pyramids. For had we found the mass of the degeneration in the anterior medium column of the right side, the posterior part of the lateral being intact, whilst on the left side a small portion only of the lateral column was diseased, it would have amounted to proof that the ordinary decussation of the anterior pyramidal fibres had not taken place.

With regard to the optic neuritis, it is noticeable that it was situated on the side opposite to the tumour, a condition observed by Dr. Hughlings-Jackson in two cases of unilateral neuritis recorded by him. And this fact would certainly appear to lend weight to the hypothesis advanced by Dr. Stephen Mackenzie, that the occurrence of optic neuritis in connection with brain disease is due rather to a diffuse cerebritis continued down the nerve than to pressure.

Another remarkable point to notice is that, notwithstanding the continued presence of a fatal lesion, the eye should recover itself and good vision return. Recovery from optic neuritis with good vision is, of course, frequently seen, but only in cases where the existing cause can be removed.

ON A CASE OF TUMOUR OF THE CEREBELLUM WITH LEFT HEMIPLEGIA.

BY CHARLES E. BEEVOR, M.B.

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THE following case was under the care of Dr. Ramskill, at the National Hospital for the Paralysed and Epileptic, Queen Square, and it is through his kindness that I am able to publish it.

F. D., a boy aged 11, was admitted into the hospital on June 22nd, 1880, and the following history was obtained from his mother.

There was no neurotic history, and he had had no previous illness of any importance.

Present illness came on five months ago rather suddenly, with pains in the head, and vomiting after everything he took. Four months ago he had a fit while in bed, and preceded by vomiting; he did not lose consciousness; the face was "drawn down on the right side," but the arms and legs were not convulsed; the face was not paralysed after the fit; he had another similar fit a week later.

About this time his sight began to be impaired, and in about another fortnight his mother noticed that his eyes were turned inwards, and they have remained so ever since. Two months ago he began to get giddy, and to reel about in his walking, and to fall forwards and to the left, and "one day he lost power" in the left arm and leg, but not completely, as he was able to walk with assistance till within a month of his admission; the left arm and leg have been getting gradually worse ever since.

His mother does not think that the face was affected the same time as the left arm and leg; but it probably was, as it was found to be affected on the left side on admission. His mother also states that he has not been able to lie on his left side for the last three months.

Present state.—Patient is a dull, heavy-looking boy, who does not take notice of anything, but answers questions slowly,

and takes a long time to do what he is told. He is very drowsy, and sleeps very much, and does not like to be disturbed.

He says he has not any pain at present, but frequently has severe pain in his forehead.

He has frequent vomiting, and the absence of food makes no difference, or very little.

Face.—On showing the upper teeth the right side of upper lip moves, and the left hardly at all; he can close both eyes equally well. Has no ptosis.

Ocular muscles.—There is internal strabismus of the right eye; he can direct either eye to the inner side, but cannot turn either eye outwards beyond the middle line; he cannot elevate or depress the eyes at all; he apparently cannot converge the eyes to a greater degree than they are already converged (?). Pupils equal, rather dilated, no reaction to light, contraction to accommodation doubtful.

Muscles of mastication not affected, and he protrudes the tongue a little to the left.

In phonation the soft palate seems to be drawn a little more to the right than left.

He swallows slowly and with difficulty.

Upper limbs.—Both wasted. Left measures about $\frac{1}{4}$ inch less than right.

Grasp of right hand = 2 kilogrammes by dynamometer.

„ left = 0 „ „

Has free movement of right arm.

With the left he cannot pick up a pin; he can just manage to raise the left hand to his head, but cannot hold it out straight in front of him; can just flex and extend the left fingers. He is unable to raise the left shoulder.

Lower limbs are much wasted, especially the left. He has free movement of the right leg, but he cannot move the left one at all, and it falls like a dead weight. No rigidity in either limb.

The power of right leg is much below normal, but this is probably due to his general weak condition.

Knee phenomenon obtained in either leg, but no excess can be made out.

Ankle clonus present in left leg, but not very well marked; absent in the right.

Plantar reflex present on both sides, but more marked on the right than the left.

Cremasteric reflex very well marked on the right side, not obtained on the left.

Abdominal and *Epigastric* reflexes very marked on the right, only just obtained on the left.

Sensibility does not seem to be affected, and he localises correctly where he is touched, on both sides all over the body.

Sight.—No diplopia; he can recognise objects—as a key—but cannot distinguish letters.

Ophthalmoscopic examination shows intense double optic neuritis, both discs very red and the margins obscured.

Hearing is much affected, a watch being heard about 12 inches away with the right ear, and only 2 inches off with the left ear.

Taste and *Smell* could not be properly ascertained, owing to the dense condition of his intellect.

Urine contains no albumen. He passes urine and fæces involuntarily into the bed.

He has almost constant priapism.

There is nothing abnormal in the heart, pulse 64.

The vomiting persisting three or four times a day, he was fed for a time on nutrient enemata only, but he still vomited every morning.

On July 15th he had a fit; he lost consciousness, and the head was drawn round to the left; there was marked arching of the back; the arms and legs were not convulsed, but became very stiff, especially the legs; the attack lasted about one minute, and in a few minutes he had another of a similar nature. He was very drowsy after them.

Patient gradually got worse and more dense and drowsy, and as far as could be ascertained he did not present any fresh symptoms; the vomiting persisted, and though he was fed with nutrient enemata he gradually sank, and died on August 4th.

His temperature on the evening before he died was 104, and just before death was 107·4.

On post-mortem examination a tumour was found shaped like a cottage loaf, measuring 3 inches long, and 2½ broad at its larger end, by which it grew from the upper surface of the cerebellum, extending on each side of its middle line. The smaller end of the tumour was free, and extended forwards between the optic thalami, and flattening the corpora quadrigemina, so that there was hardly any trace of them visible; the right optic thalamus for about the inner ¼ of an inch, the right crus cerebri, and the right superior cerebellar peduncle, were also compressed; there was some thickening of the meninges about the tumour, but there was no sign of pressure on the pons, medulla, or any of the cranial nerves.

In this case there were the following symptoms met with in intra-cranial tumours: pain in the head, daily purposeless vomiting, and double optic neuritis.

The localisation of the tumour to the cerebellum was not warranted by the symptoms as they first presented themselves,

as during the first month he only had pain in the head and constant vomiting. The description of the fits as given by his mother was very imperfect, and she did not know very much about them, but those he had in the hospital were cerebellar in character, arching of the back with tonic rigidity of the legs, and absence of clonic convulsions. The first fit occurred about a month after onset, and about this time his sight began to fail; but whether this was due to the optic neuritis is doubtful, as Dr. Hughlings-Jackson has shown that double optic neuritis may exist and yet with no impairment of vision.

In one and a half to two months after onset of illness he was one day noticed to have internal strabismus, and he gradually became giddy and staggering in his walking, falling forwards and to the left, and he suddenly one day lost some power in the left arm and leg.

It seems probable that the tumour, which had hitherto kept to the middle line of the cerebellum and had produced no symptoms pointing to its position, now began to grow forwards and to the right, and press on the right crus cerebri; for it has been found that lesions of the cerebellum *per se* do not cause hemiplegia. The loss of power in the limbs was not complete, but gradually increased as the pressure of the growth on the crus became more and more; the leg being affected, on admission, more than the arm, and so differing from ordinary cases of hemiplegia, and thus bearing out Dr. Bastian's opinion (Paralysis from Brain Disease) that these cases are due often to pressure on the pons Varolii from lesions of the cerebellum.

With regard to the strabismus and paralysis of the movements of the eyeballs, they were probably due to the compression of the corpora quadrigemina, which are now considered to be the centres for the co-ordination of the movements of the eyeballs; for in this case there was no paralysis due to lesion of any one nerve, for the portions of the third nerves supplying the internal recti were not affected, while the parts of the nerves supplying the superior and inferior recti were paralysed, clearly showing that it was not the nuclei or the nerve trunks themselves that were affected, but a centre higher up, which co-ordinates the movements of the eyes in different directions; and this case would seem to bear out the theory that the centres for converging the eyes, and for elevating and depressing them were paralysed, while the centre for causing lateral movement was very much impaired.

The corpora quadrigemina are not now considered the seat of vision, and in this case, though they were so much com-

pressed, the patient was able to see well enough to distinguish objects.

The pupils were immovable, and this has been found in other cases to be associated with bilateral lesion of the corpora quadrigemina.

With regard to the situation of the tumour in the median lobe of the cerebellum, there was one symptom which Dr. Bastian (Paralysis from Brain Disease) has stated to be met with most commonly in lesion of this part, viz., persistent priapism, which occurred the whole time he was in the hospital.

The superficial reflexes in this case were altered as they frequently are in hemiplegia, viz., diminished on the paralysed side; the plantar, abdominal and epigastric reflexes being less marked on the left than on the right, while the left cremasteric was absent, the right being very active; on the other hand, ankle clonus was obtained on the left side, but absent on the right—no difference could be made out between the knee phenomena of the two sides.

This diminution of the superficial reflexes on the paralysed side has been explained by a theory based on Setschenow's experiment, who stimulated the optic lobes in a frog and thereby found that reflex action was not so active, the optic lobes exercising an inhibitory action over the reflexes. In man it has been supposed that the optic thalami exercise this inhibitory power over the superficial reflexes; but normally the thalami are themselves inhibited by the cerebral hemispheres, so that when hemiplegia occurs from a lesion cutting the connection between the hemisphere and the optic thalamus, the latter has full play and inhibits the superficial reflexes of the paralysed side.

In this case, however, the superficial reflexes were diminished on the paralysed side, the connection between the optic thalamus and the hemisphere was intact, and the lesion was situated below the optic thalamus and between it and the spinal cord, so, in the first place, the optic thalamus was still inhibited by the hemisphere; and, secondly, the spinal centres were cut off from the influence of the thalamus.

Besides, if this theory were a correct explanation, the superficial reflexes ought to be diminished directly the inhibitory action of the hemisphere is removed—as by a sudden hemorrhage—and at the onset of the attack; but this is not found to be the case, the superficial reflexes not being absent or diminished on the paralysed side till some time after an apoplectic seizure.

The staggering in his walking and the falling forwards and towards the left side, which occurred about six weeks or two

months after the onset of his illness, were probably due to pressure of the tumour on the right half of the cerebellum, and on the right superior cerebellar peduncle.

The right half of the cerebellum works with the left half of the cerebral hemispheres, and they both govern the right half of the body, and when one half of the cerebellum is pressed upon by a tumour, the opposite half over compensates and carries the body over to the opposite side (Dr. Ferrier), and in this case, with a lesion of the right half of the cerebellum, the body was carried over to the left side. Falling forwards has also been considered as occurring in lesions of the anterior part of the median lobe of the cerebellum.

The power of hearing was very much affected, and on the left side more than on the right, which might have been caused by the pressure on the right crus cerebri involving the fibres passing up from the auditory nucleus to the higher centres for hearing in the cerebral convolutions. Hearing on the right side being affected would probably be due to the deficient condition of the patient's intellect; but, besides this, the theory has been advanced as to the possibility of the auditory nerves suffering from neuritis, from intracranial pressure, in a manner similar to the optic nerves, and thus their functions being impaired.

The involuntary evacuation of excreta—which is rare in young cases of hemiplegia—could be accounted for by the patient's mental condition, as he did not take notice of anything, and only spoke when asked a question.

For the appended account of the microscopical appearances of the tumour I am very much indebted to Mr. Victor Horsley, assistant pathologist at University College, who very kindly examined the growth for me.

Tumour of Cerebellum.

The growth in this case evidently sprang from the under surface of the dura mater, extending inwards towards the brain. Throughout it was soft and greyish in colour, obviously cystic in the older portions and more uniform in texture towards the growing edge. Microscopical examination showed it to consist of a fibrous stroma and two classes of corpuscular elements.

The stroma growing from the dura mater was composed of dense fibrous tissue at the periphery, but soon divided into more delicate trabeculæ consisting of fibres and spindle-cells, while here and there patches of small round cells, forming a loose embryonic tissue, were found in the more actively growing portions. In several places the stroma was evidently

myxomatous, while everywhere the fibrous trabeculæ formed alveoli containing the round cells described below, and sometimes presenting even the appearance of an alveolar sarcoma. Besides the connective tissue corpuscles of the stroma, there were two distinct classes of cells which formed the main mass of the tumour.

1. Round corpuscles. These were large round or oval cells with granular protoplasm, a large nucleus, and bright nucleolus, and measuring about $\frac{1}{1000}$th inch in diameter. These occupied the alveolar spaces in the stroma sometimes in large masses, at others the cells would be enclosed in little single meshes.

2. The second class of corpuscles constituted an epithelial growth extending through the tumour in different directions. In its earliest stage it consisted of columns of cells which at the periphery were columnar in shape, but towards the centre of the column passed through the transitional stages until the central cells were large flat epithelial plates (figured by Cornil and Ranvier, part 1, p. 134). In other cases the cells in the centre of the column had undergone extensive colloidal change, and this had evidently led to the production of the cysts described below. The epithelial corpuscles were also scattered in groups through the tissue of the stroma without apparently any columnar arrangement, and were perfectly distinct from the large round cells occupying the alveoli of the stroma, since the latter spaces were bounded by well-marked trabeculæ, while the epithelial columns were always found where the stroma was myxomatous.

An additional point of interest in the tumour was the formation of cysts. These cysts were of various sizes up to 2 mm. in diameter, and situated in the older portions of the growth, containing either simply granular débris, colloid material or cells which were not completely degenerated.

No relationship could be detected between the blood-vessels and epithelial columns, and the groups of epithelial cells had not become calcified.

The growth is obviously that described by Cornil and Rauvier under the name of "Angiolithic sarcoma," by Virchow as "psammoma," and Rindfleisch as "Epithelioma myxomatodes psammosum." Neither Cornil and Ranvier nor Rindfleisch mention a round-celled growth which was so striking a feature in this case.

The spinal cord on examination proved to be healthy throughout.

SPASMODIC WRY-NECK; EXCISION OF A PORTION OF THE SPINAL ACCESSORY NERVE—DEATH FROM ERYSIPELAS.

BY WALTER RIVINGTON, F.R.C.S.

Surgeon to the London Hospital.

HENRY H., 58, was admitted into the London Hospital in February 1875, under Dr. Hughlings-Jackson. The notes of the case were taken by Mr. Charles Mercier.

The patient had the ordinary form of spasmodic wry-neck, and had suffered about eighteen months. He was a healthy-looking man, and said that he had had no serious illness except small-pox when a child. He never had rheumatic fever or scarlet fever. He did not know that any of his blood-relatives had suffered from any kind of nervous affection. Detailed inquiries were made in this regard, and his statement must of course be taken for what it is worth. Some years ago the patient, who had been a weaver, suffered from what we may call "weavers' cramp," by which is meant a condition analogous to "writers' cramp." The following account of it was obtained by Mr. Richard Atkinson, on the patient's second admission, but is conveniently narrated here as part of his history. It was no part of the case, in so far as association with the wry-neck goes, but it may possibly be supposed by some to show a neurotic tendency: "In weaving, the operator throws the shuttle, to which the warp is attached, through the woof, and then catches it with the other hand. He then performs the same motion in the reverse order, so that the shuttle is brought back to the hand from which it started. In throwing the shuttle it is held between the thumb and middle finger, the index-finger resting on it behind. The propulsion is effected by a slight flexion of the wrist together with a flexion of the index-finger. The patient performed exactly the same motions with his right hand as with his left, but he never got the cramp in his right hand, only in his left. The cramp came on thus: Having thrown the shuttle from

his left hand, he would carry the wrist backward so as to be ready to catch it again when returned from the right hand, and it was after throwing the shuttle that the cramp seized his hand so as to overflex the fingers and prevent him being ready to catch the shuttle on its return from his right hand. Great accuracy and rapidity of movement being requisite for weaving purposes, the patient was thus on several occasions unable to follow his trade. He took to another and simpler method, and for some years he succeeded in this; but again the cramp seized him and, as before, the left was the hand affected. In this method he used his left hand only to push a beam, called a batten, backwards and forwards. The right hand threw the shuttle through the woof; instead of the left hand catching and returning it there is a box which does this. The movement seems to have consisted of an alternate slight flexion and extension of the elbow, the wrist being kept a little extended. Though there would be little or no movement of flexion and extension performed by the wrist, yet there would be considerable strain on the flexors of the fingers. In this method of weaving, as in the previously described, the cramp consisted of an over-flexion of the left wrist, so that he became unable properly to remove his batten." The following is Mr. Mercier's note of the case in February 1875: "He first noticed the onset of this complaint eighteen months ago. At that time he had an aching pain at the sides of his neck (he indicates the upper portion of the sterno-mastoids), equally bad on both sides. It was just as if he had sat in a draught and caught cold. It was not persistent: was worse when he turned his head in either direction, and went off if he kept his head for some time still in the middle line. It was always absent at night. Afterwards he had a pain at the back of the head on a level with the occiput, on the right side only, an aching pain. This pain has never been on the left side. He had had the pain for six months before the head began to move. At first the movement was very slight indeed; when he imitates it, it is scarcely perceptible, and as he reproduces it, it is a simple rotation from left to right, without any lateral or oblique movement. He could get it back by effort, but it immediately returned, not more strongly, however, for the check it had received. At this early date it never twisted the head round even after repeated spasms. It very gradually got worse and worse, with an occasional remission for a day or two. At first the spasm ceased directly he put his head on the pillow, but gradually he began to have it at this time also. His wife does not know whether he moves his head during sleep. He says that his head is moving when he wakes in the morning. He has always had pain like a cramp in the back

of the neck whenever he moves his head round much. At the present time the movements are usually a slight rotation of the head towards the right, while at the same time it leans down towards the right shoulder, and the chin is raised. During the movement the left sterno-mastoid becomes extremely hard, and the left trapezius hardens slightly. At times the rotation is very much greater, and the head leans much more over the shoulder, the chin is also raised more, but not so much more. In these more extensive movements the left sterno-mastoid is quite rigid, and the left trapezius is also brought strongly into action. In the return movements, the corresponding muscles on the opposite side contract. The movements consist of a rapid succession of small ones scarcely abrupt enough to be called jerks, but yet distinctly separate."

November 1*st*, 1875.—Patient has been attending on and off ever since. He wore an apparatus with benefit for some time, but the torsion has become so bad that it is no longer useful. He is thinner, and has done no work for twelve months. He was re-admitted on January 21st, 1876.

No kind of treatment did the man any good excepting the temporary benefit from the apparatus. Dr. Hughlings-Jackson consulted Mr. Rivington as to the propriety of dividing the spinal accessory nerve, and, after consultation, advised the patient to have the operation performed. He was transferred to Mr. Rivington's care, whose further report concludes the case.

"The patient was a man of slender build with a feeble circulation. The heart-sounds were normal but weak; breath-sounds were feeble, expiration being prolonged. Urine was free from albumen.

"The operation was performed on February 7th, after the method adopted by Mr. Campbell de Morgan. The patient having been anæsthetised, an incision was made along the posterior border of the left sterno-mastoid for 2 or 3 inches. The skin superficial fascia and platysma were divided successively. The external jugular vein, which was in relief from distension with blood, was carefully protected and drawn aside. The deep cervical fascia was then cautiously incised, and the great auricular nerve was recognised ascending towards the lobule of the ear, and near this the spinal accessory was found. The nerve was then traced through the substance of the sterno-mastoid, and a piece was excised from the trunk before its entrance into the muscle. Galvanism was employed to verify the nerve, the sterno-mastoid being thrown into strong contraction when the lower part of the divided nerve was included in the circuit. One small vessel was ligatured, and there was some oozing during the performance of the operation. The

edges of the wound were brought together with wire sutures, pads of lint and strapping being applied, and a bandage over all. After the effects of the chloroform had passed off, the neck was examined. The spasms and twisting of the neck still continued. These were found to be caused not by the left sterno-mastoid, which was quite relaxed, but by the action of the muscles at the back of the neck on the right side. The right splenius was tense. The head was twisted quite as much as before the operation, the face being turned round to the right side, and the chin tilted upwards. As it was evident that nothing more could be done, the patient was sent to bed. He had a dose of chloral hydrate in the evening and slept for some hours. Early the next morning the patient shivered a good deal. His tongue was dry and furred. Pulse was 120, moderately strong, and the temperature stood at 103°·2. The spasms continued intermittently.

"*Feb. 9th.*—Patient slept again last night pretty well after chloral hydrate. Tongue slightly furred. Appetite fair. Bowels inactive. Pulse 90 and moderately strong. Skin moist. Wound clean. Wound dressed by Mr. Mears (the dresser to the case, from whose notes most of these particulars are extracted). Wound looking clean; very little discharge; no tendency to bag; edges seem united save at one spot. Wet lint applied. Movement of head nearly stopped; scarcely if any jerking.

"10*th.*—Wound healthy; edges united in parts; highest stitches taken out; no bagging, pulse 84, temp. 99°·2. Head quiet.

"11*th.*—Head still quiet; bowels inactive; tongue slightly furred; appetite moderate, pulse 98·2. Wound healthy; patient slept well after chloral hydrate.

"12*th.*—Patient says he is better; tongue cleaner; bowels have acted. Pulse 84, good, temp. 98°·2. Remaining stitches removed; wound united in the middle; head quieter.

"14*th.*—Not much sleep last night on account of pain at back of head. Appetite good. Pulse 90, fairly strong; temp. 99°. Wound very healthy, moderate discharge of healthy pus, granulating rapidly.

"16*th.*—Temperature 103°·2 in the morning, no apparent cause for the rise. Patient otherwise in good condition.

"17*th.*—Discharge slight, health good; temp. 193°. Head quiet.

"18*th.*—Wound not quite so healthy, skin dry, scaly and purplish, no erysipelatous tint; secretion of a dirty brown colour and scanty. Patient sleeps well and has a fair appetite, temp. 98°·4; pulse regular, 96; bowels active.

"19*th.*—Patient restless all night in spite of hyd. chlor.

draught. Tongue dry and dark; pulse 108, sharp and rather weaker; skin moist; temp. 100°·2.

"21*st*.—Erysipelas appeared yesterday, temp. 103°; pulse 112; lot. plumbi evaporans ordered. To-day the erysipelas has spread over the scalp, left cheek, neck and shoulder. Temp. 104°; pulse 102, full; tongue brown and dry, skin hot and moist; no appetite; dirty pus scantily secreted.

"22*nd*.—Patient slept a little. Removed to Erysipelas ward, and ordered quin. sulph. and fer. perchlor. Skin moist; temp. 103°; no secretion from wound, but a yellow crust on the surface. Erysipelas has spread right over the scalp, left cheek and eye, left side of neck, and upper part of thorax; in some places blebs have formed.

"23*rd*.—Erysipelas has invaded opposite side of neck and has become deeper seated; bulloe everywhere. Pulse 99, full; temp. 101·3°. Takes a great deal of milk, nothing else. Skin painted with arg. nit.

"24*th*.—Very restless and delirious all night. Face greatly swollen on both sides, also his neck. Wound is quite dry; small slough is forming at inner angle of left eye. Pulse 160 and very weak; temp. not taken. Towards the afternoon the erysipelas subsided a good deal, but patient continued delirious. He was very suspicious and excited and obstinate. He would sit up in bed resting his head, which was quite straight and quiet, on his right hand and talk loudly. Once he declared that his medical attendants were trying to keep his case to themselves, but they would not be able to do it. Everybody would hear of it. There were no symptoms of arachnitis.

"25*th*.—This morning the swelling is again present, together with some dusky redness. The right upper eyelid contains a good deal of pus. When the patient moves there is a great deal of spasm and twitching of the muscles. Patient very apathetic. Pulse weak and not rapid.

"26*th*.—About the same; unable to swallow; fed with enemas. Bowels opened unconsciously; no convulsions. Patient can now move his arms but slightly and jerkingly, and grasps feebly. Lies quiet. Pulse very weak and irregular; temp. not taken. Tongue very dry, hard, brown and cracked."

This is the last note made by Mr. Mears, who paid great attention to the case throughout. Death took place a few hours later. The post-mortem examination was conducted by Dr. Sutton. The visceral appearances were those due to congestion from the erysipelas, and did not present anything to account for the result of the case.

The brain and spinal cord were carefully examined by Dr. Jackson, Dr. Sutton, and Mr. Rivington, and were reserved by

Dr. Sutton for microscopic examination. Unfortunately the specimen seems to have been mislaid, for no note could ever be discovered concerning it. From the firmness of the spinal cord in the cervical region, both in its entirety and when cut into slices, Mr. Rivington suggested that it was sclerosed, and Dr. Sutton thought this very probable. The ends of the spinal accessory nerve were found separated, and had been completely divided.

Remarks by Mr. Rivington.—My reasons for following Mr. De Morgan's mode of operation were that it afforded more room to the operator than seeking for the nerve in front of the sterno-mastoid, and seemed less likely to implicate the jugular vein. I performed both operations previously on the cadaver, and found that I could easily reach the spinal accessory and divide it in front of the sterno-mastoid, but that I should be obliged to work in a hole, and if any troublesome bleeding occurred from the sterno-mastoid branch of the occipital artery, it would probably be necessary to enlarge the incision and divide the external jugular vein, &c., before the artery could be comfortably tied or twisted. By Mr. De Morgan's method no important nerve or vessel need be touched, and it was quite easy to trace the spinal accessory and isolate sufficiently for the purpose. The termination of the case was disappointing. The operation was not performed under the carbolic spray, as it did not seem to be necessary, and would have tended to obscure the dissection. There had been a few scattered cases of erysipelas in the hospital, but none in the part of the building in which the patient was located. By what channel he became affected I could not determine. In reference to this matter it has occurred to me to put the question whether, assuming the cervical cord to have been the subject of sclerosis, a predisposition to vascular disturbance from slight irritation had not been created. Seeing that the spinal accessory nerve, and indeed the other cervical nerves, had become the channel for the transmission of irregular impulses due to the central disease, and set going perhaps by any slight peripheral impressions, might not a *small* amount of irritation in the wound, and a *small* quantity of peccant matter in the blood, cause abnormal disturbance to the vaso-motor centre and vaso-motor paths in the cervical portion of the spinal cord? The erysipelas spread no further than the head and neck, and shoulder and upper part of the thorax—over the regions covered by the nerve-supply from the cervical plexus and cranial nerves arising from the medulla, and inasmuch as the reflex connection between sensory and vaso-motor nerves is very intimate, it appears to me that a good deal of the vascular disturbance may be accounted for in this manner. Admitting that some

materies morbi had entered the blood through the wound, I cannot help thinking that but for the neurosis the patient would have recovered, for many cases worse than his get perfectly well, and deaths from erysipelas have not been by any means frequent in my experience at the hospital. As the spasms noticed after the operation completely subsided in a few days, and did not recur, it is fair to assume that he would have been relieved of his complaint if he had recovered from the erysipelas."

Remarks by Dr. Hughlings-Jackson.—"It is worthy of note that the erysipelas did not set in till nearly a fortnight after the operation, when the wound had nearly healed. The patient shivered a good deal the morning after the operation, and the temperature stood at 103°·2. Does the interval between the shivering and the appearance of the erysipelas represent a stage of incubation?"

Abstracts of British and Foreign Journals.

Kirchoff on Cerebral Glosso-pharyngo-labial Paralysis. (*Archiv f. Psych.* Bd. xi. p. 132.)—A man, aged 24, while perspiring profusely, stepped into water for a bathe. Scarcely had he done so, when he became giddy, had convulsive tremors in his extremities, lost the power of speech, and was unable to swallow. Saliva flowed from his mouth, and his face was drawn to the left. These symptoms disappeared in a few days, but a week later he had a similar attack. On admission into hospital, July 29, 1877, it was observed that he articulated with difficulty; labials and gutturals especially were troublesome; linguals he spoke with comparative ease. The lips were moved little in speaking; he could not whistle, but was able to approximate the lips. Saliva flowed from the mouth, and there was excessive secretion of tears. The tongue was not protrusible more than 1 centimetre from the mouth, and it was moved clumsily in the act of biting. At the time of examination swallowing was unimpeded, but the glottis was closed tardily. Patient often laughed without occasion. There was disease of the mitral valve.

During the progress of the case there were marked remissions and exacerbations. On November 2 swallowing was only possible with the head bent back, and even then was difficult. Saliva flowed from the mouth continually, and in his laugh, crowing and whistling sounds were often heard. His arms were shaky. On December 25 his face was suddenly drawn to the right, and his left arm and leg became powerless. Chronic convulsions occurred from time to time up to his death, on Jan. 5.

The post-mortem revealed embolic softening of the posterior two-thirds of the right corpus striatum (caudate nucleus?), the underlying internal capsule, the outer segment of the lenticular nucleus, the claustrum, external capsule, and island of Reil. The focus of softening in the lenticular nucleus was distinguished from the other softened portions by being surrounded by a wall of compact sclerosed tissue. Very careful microscopic examination failed

to show any disease of the medulla or pons. The author attributes the glosso-labial paralysis to the lesion of the lenticular nucleus; and the hemiplegia to the quite recent lesion of the caudate nucleus, internal capsula and other parts. Cases of bilateral affection of the face, tongue, and throat, caused by unilateral lesion of the cerebrum, are rare; the author cites two, recorded by Lépine and Magnus respectively.

Virchow on Progressive Facial Hemi-atrophy.—Virchow (*Berliner klin. Wochenschr.* 1880, No. 29, and abstract by Bernhardt in *Centralbl. f. med. Wissensch.* 1881, No. 3) reports two cases of this disease under the name of "neurotic atrophy." The first was a man, aged 42, whose face became atrophied on the left side in his ninth year. Virchow had seen the same patient twenty-one years previously, during which time the disease had made no appreciable progress. The second case was a woman, aged 41, in whom the disease had shown itself sixteen years ago, after an inflammatory affection of the skin of the left eye. Shortly before this the patient had fallen on the back of the head, and during childhood had often suffered from furunculus. There was atrophy not only of the left side of the face, but of a large area extending from the middle line of the back (between the fourth and seventh dorsal vertebræ) outwards over the left infra-spinous fossa to the shoulder, and thence down the back of the arm to the ulnar side of the forearm. The area of distribution of the left radial nerve was markedly atrophied. Tactile sensibility was normal; at one time sharp twinges of pain were felt in the eye and arm, but at the time of examination these had given place to a numb feeling in the arm and a feeling of cold in the little finger. The author regards the disease, in its origin at least, as a peripheral affection of the nerves. The morbid process, inflammatory or otherwise, may spread upwards till it reaches the ganglia in the basis cerebri or spinal column. The atrophic areas correspond to the course of various nerves, but not to the whole area of distribution of these nerves; certain fibres only of the nerves become diseased. The author remarks that there are few things in nerve pathology more striking than the unequal affection of the fibres of the same nerve in this disease. He regards lesion of the trigeminus as the starting point of the malady. The bones atrophy if the affection commences in the earlier years of life.

Flascher (*Berliner klin. Wochenschr.* 1880, No. 31) describes a case of *bilateral* facial atrophy, occurring in a woman aged 23.

When an infant she fell and injured her forehead, and shortly afterwards had an attack of measles, without eruption. It was soon after this that the facial atrophy appeared. The trigeminus, as a whole, was not implicated, but only particular branches of it, and only particular fibres of these branches. In the atrophic places the vaso-motor system was unimpaired, tactile sensibility diminished, the secretion of sweat paralysed. The bones were atrophied, so also the facial muscles and the masseters and temporals. There was external strabismus of the left eye, the pupil of which was irregular, widely dilated, and non-reactile; sight was defective, and the optic disc atrophied and white. The weakness of sight and strabismus were detected about the same time as the facial atrophy. Flascher, too, regards the disease as a peripheral affection of the nerves, coming on in this case as a sequela of measles.

Hammond (*Journ. Nerv. and Ment. Disease*, April, 1880) observed a case of progressive facial atrophy in a girl, aged 14. The affection was of gradual growth, and did not attract attention till she was 12. There was a decided difference in the size of the two sides of the face. On the left side there were three depressions, two near the angle of the mouth, and one above and slightly in front of the ear. The left half of the tongue was much smaller than the right, and the palatine arch was flatter on the left than on the right side. Muscles supplied by the motor branch of the fifth nerve, by the facial and by the ninth, were atrophied; microscopic examination of them showed atrophy without degeneration. There was no appearance of paralysis. The skin, hairbulbs, cellular tissue, and even the bone (temporal) were also atrophied. There were occasional paroxysms of numbness lasting a few minutes in the left side of the face. Defective sensibility was found only in a small region over the left half of the orbicularis oris. The author considers the nuclei of the trigeminus, facial, and hypoglossal as the primary seat of the disease, which consists essentially in lesion of the trophic cells of these nuclei.

Eulenburg on Facial Spasm. (*Centralbl. f. Nervenheilk.* 1880, No. 7, and *Cbl. f. med. Wissensch.* 1880, No. 47.)—A woman, aged 25, after an attack of transient aphasia, without loss of consciousness, became subject to violent and painful contractions of the muscles of the left face, especially of the orbicularis palpebrarum. In severe attacks the convulsions spread to the muscles supplied by the left spinal accessory, and to the flexors of the left hand and

digits, and on rare occasions to the right side of the face and neck. There was hyperalgesia of the left face, and numerous painful points were found along the course of the trigeminal and facial nerves. Galvanisation, bromide of potassium, injections of atropine, morphine and curare, and neurotomy of the supra-orbital nerve having proved ineffectual, the facial nerve was stretched. The operation cured the spasm, but left almost total paralysis of the facial nerve. The muscles of the underlip were only partially paralysed, and those supplied by the posterior auricular nerve were quite intact. A curious result was the abolition of the sense of taste in the anterior half of the left side of the tongue; sensibility and salivary secretion were normal. After a time spontaneous, intermittent fibrillary contractions, tonic-clonic in character, appeared in the depression of the lower lip, which soon became the seat of a contracture. Fifteen weeks after the operation, the sense of taste returned. As lesion of the chorda tympani is out of the question in this case, Eulenburg attributes the loss of taste to lesion of a filament, which, according to anatomists, is sometimes found connecting the peripheral branches of the glossopharyngeal and facial nerves.

Mossdorf on Aphthongia.—This was the name given by Fleury in 1865 to a variety of aphasia, characterised by spasm of the muscles supplied by the hypoglossal nerve whenever the patient attempts to speak. In Mossdorf's case (*Cbl. f. Nervenheilk.* 1880, No. 1, and *Arch. de Neurologie*, 1880, No. 2), on the patient, a boy aged 17, attempting to speak, the hyoid and abdominal muscles became cramped, respiration ceased, and through the half-opened mouth the tongue was seen contracted, its tip firmly pressed against the lower incisors, and its dorsum against the palate. In a few moments the abdominal muscles relaxed, and respiration was resumed, but the spasm of the tongue and hyoid muscles persisted. The movements of the tongue, lips, and face were, with this exception, quite normal. Latterly the boy was unable to reply to his parents, or even read aloud when alone. The difficulty of speech showed itself when he was six years old, and was attributed to fright. A cure was effected by galvanisation; the negative pole was placed in the nape of the neck, and the positive pole slowly moved up and down the spine.

Schultze on Multiple Cerebro-Spinal Sclerosis and General Paralysis.—Schultze (*Arch. f. Psych.* Bd. xi. p. 216) relates a case

which, during the first two years of its course, was looked upon as cerebro-spinal sclerosis. The symptoms were as follows: Tremors of the extremities during voluntary movements, paresis of the legs and of the right arm, pains in the extremities, impaired sensibility of the right arm, at times incontinence of urine, hallucinations of sight, diplopia, vertiginous seizures, epileptiform attacks, slow, stuttering, monotonous speech. There were marked remissions in the symptoms, and twice the gait of the patient, who, we may mention, had suffered from syphilis, so far improved that he was discharged. On his third admission into hospital, mental disturbances for the first time showed themselves. He became acutely maniacal, had grandiose ideas, and presented up to the time of his death the typical symptoms of general paralysis. It is a remarkable fact that tremors were never observed during voluntary movements after the appearance of the mental disorder. On *post-mortem* examination there was found, in addition to the usual accompaniments of general paralysis (diffuse hyperplasia of the connective tissue and atrophy of the cerebrum), multiple sclerosis of the cord and diffuse sclerosis of the brain. In another case of general paralysis, the author found small patches of sclerosis in the cord, and diffuse sclerosis in both cord and cerebrum.

The concurrence of the two diseases, multiple sclerosis and general paralysis, rare though it seems to be (a case by Claus is reported in 'BRAIN,' Vol. II. p. 142), is only what might be expected from their pathology, for they are both characterised by a widespread hypertrophy of the neuroglia with the formation of granule-cells. Sclerotic changes in the cord, particularly in Gall's columns, and the pyramidal strands occur frequently in general paralysis.

The first case offers a few subsidiary points of interest:—(1.) the cord in the fresh state appeared quite normal; it was only after immersion in Müller's fluid that the sclerosed patches became visible. (2.) Though the whole area of the posterior columns was sclerosed in the lower part of the dorsal region, and again in the lower part of the cervical enlargement, there was but slight secondary degeneration of Goll's columns. The lateral pyramidal strands were degenerated through the greater part of the cord, but in the upper cervical region were quite intact, which shows that the degeneration was of spinal origin. The relation of multiple sclerosis to secondary degenerations is still far from clear. Schultze suggests that the pyramidal strands and Goll's columns are, from their anatomical constitution, the parts that suffer most severely in general hyperplasia of the connective tissue of the

cord. (3.) Corresponding to the isolated atrophy of the interossei muscles of the right hand, there was lesion of the right anterior cornu in the lower part of the cervical enlargement, which coincides with other observations bearing on the localisation of the spinal centres for the small muscles of the hand.

Seeligmüller and Schmid on Hereditary Ataxia.—Seeligmüller relates the histories of two brothers who suffered from this disease. The younger, aged 26, showed symptoms of ataxia of the lower extremities when 12 years old. Ten years later, nystagmus developed, and quite recently slight ataxia of the superior extremities had been observed. The ataxia of the legs was visible both in walking and in standing with the feet together (static ataxia). The movements of the eyes had not the usual rythmical character, but were more irregular and jerking, and corresponded with Friedreich's atactic nystagmus. There were no fibrilly contractions, except slight ones in the protruded tongue; sensibility and electrical irritability of nerve and muscle were normal; there were no paralyses, contractures, or atrophies. The secretion of saliva was abnormally abundant. This case differs from those described by Friedreich in the following points: (1) There was no ataxia of speech; (2) the patellar reflexes were heightened, not absent; (3) there were cerebral symptoms, in the shape of psychical manifestations, *e.g.* forgetfulness, tendency to reverie, perverted sexual instincts, and melancholia when 16 years old; and (4) there was occasional impairment of the vesical and anal sphincters. In the elder brother, aged 28, the nystagmus and the static and locomotor ataxia of the lower extremities were less marked. The upper extremities were unaffected. The patellar reflex was lively, sensibility intact. The patient was forgetful and absent in mind; he spoke very rapidly, but sometimes not very intelligibly, as he would hurry from sentence to sentence without finishing them. Father and mother were cousins, and nervous diseases were common in the family.

Schmid's case (*Centralblatt f. med. Wissensch.* 1880, No. 25) was a boy of 18, whose gait five years before had become uncertain and peculiarly rapid, and at length resembled that of a drunken man. There was marked locomotor and static ataxia of the lower extremities, and slight incoordination in the movement of the arms. Closing the eyes, or being in darkness, did not intensify these symptoms. Muscular power and electrical irritability were normal, the superficial reflexes unaffected, the patellar reflex

absent. Speech was halting and inarticulate, and the eyes exhibited nystagmic movements. Sensibility was intact, and the sphincters acted naturally. There were no mental symptoms. At a later period the patient became unable to walk, and slight sensory disturbances were noticed in the lower extremities. A younger brother, when 11 years old, began to show symptoms of locomotor ataxia, and his patellar reflex disappeared. A sister, aged 9, is healthy up to the present. There was no special tendency to neuroses in the family.

Remak on Localised Ataxia and Ephidrosis. (*Berliner klin. Wochenschr.* 1880, No. 22, and *Centralbl. f. med. Wissensch.* 1880, No. 51.)—A man aged 38, infected with syphilis twelve years previously, had suffered for five years from gradually increasing anæsthesia of the right forearm, hand, and fingers. The movements of the limb were in a high degree ataxic, but not diminished in strength. Shortly after the appearance of the ataxia, excessive secretion of sweat was observed on the right half of the head, and slight reddening of the face. There was myosis of the left eye. Lately symptoms had developed which suggested commencing tabes—unsteadiness of gait in darkness, or when eyes were shut, a feeling of numbness in right sole, difficulty in micturition, absence of patellar reflex. The author diagnosed a patch of sclerosis in the right posterior half of the cervical enlargement, with secondary changes in both posterior columns. The unilateral ephidrosis may be due to lesion of the cerebro-spinal secretory centres, or perhaps to lesion of the cervical sympathetic.

W. J. Dodds, M.D., B.Sc.

Gaucher on the Morbid Anatomy of Diphtheritic Paralysis.—Gaucher (*Journal de l'Anatomie*, No. 1, 1881) reports the result of an investigation of two cases of Diphtheritic Paralysis. In one of these no lesion was discoverable, either in the muscular or nervous system. In the second, however, there were well-marked appearances corresponding in most points with those described by Déjerine as characteristic of this form of paralysis. Déjerine had found that the essential lesion was a parenchymatous neuritis of the anterior roots of the spinal cord, characterised by a segmentation of the myeline and multiplication of the nuclei in the sheath of Schwann with complete or partial disappearance of the axis cylinder. Further, in the anterior horns, the cells were less numerous, rounded and deprived of their processes.

Gaucher concludes from his own researches that there are never lesions of the muscular system, but that the lesions are confined to the nervous system, which, however, are not constant.

Apart from general congestion of the nervous centres which is not characteristic, these lesions are specially marked in the anterior roots, but generally only a certain proportion of the nerve tubes are implicated. The lesions are total disappearance of the myeline and considerable proliferation and enlargement of the nuclei of the sheath of Schwann.

The axis cylinder, however, is continuous throughout, distinct and without alteration. The neurilemma is free from lesion.

Morgan and Dreschfeld on Idiopathic Lateral Sclerosis.—In the *British Med. Journal*, January 29, 1881, Dr. Morgan relates the clinical history of three cases of so-called "spasmodic spinal paralysis" (Erb), or "tabes dorsal spasmodique" (Charcot). In one of the cases which died, an examination of the spinal cord was made by Dreschfeld, who found the following morbid appearances. The cord examined in the fresh condition, ten hours after death, presented no abnormality, except softening in the lower dorsal region. After hardening in bichromate of ammonia, sections of the cord showed light-coloured patches in each lateral column, specially marked in the dorsal region and less so in the cervical and lumbar regions. After staining with carmine the sclerosed patches were seen in the cervical region to be in close relation internally with the grey matter; anteriorly they did not extend up to the anterior horns, and externally did not reach the surface of the white substance. The grey matter of the anterior and posterior horns was normal. The patches in the dorsal region were more distinct and extended further anteriorly. In the lumbar region the sclerosed patches were situated close to the outer side of the posterior horns, extending almost up to the outer border of the lateral columns posteriorly, but only a little way in an anterior direction. The grey matter here, as in the cervical and dorsal regions, was normal.

Some of the microscopic sections were submitted to M. Charcot, who characterises this case as the first in which sclerosis has been demonstrated in the lateral columns without implication of the grey matter or posterior columns.

Dalton on the Visual Centres.—Dr. J. C. Dalton (*The Medical Record*, March 26, 1881) gives the details of two experiments on

dogs, with reference to the localisation of a visual centre in the cortex.

In the first experiment the cortex was destroyed at a point corresponding with the bend of the second external convolution, and, as was found post mortem, extending also into the two convolutions situated between this and the fissure of Sylvius. As the result of this lesion there was complete blindness of the opposite eye, with apparently normal vision in the eye of the same side. Motor power and general sensibility were unaltered. The animal died on the third day from encephalitis.

In the second experiment the lesion was more accurately confined to the second external or angular convolution, extending only partly into the convolution above and behind it. As the result of this lesion there was blindness of the opposite eye, and this only. The animal lived for twelve days, continuing blind in the eye opposite the lesion to the end.

The only particulars in which these results differ from those of Ferrier on monkeys, is their persistence, in regard to which he makes some suggestions and criticisms.

But the evidence he says warrants the following conclusions:—

1. Extirpation of the angular convolution causes loss of visual perception on the opposite side.

2. This operation is not followed by any disturbance of the intelligence, attitude, power of locomotion or general sensibility.

3. It does not interfere with the local sensibility of the retina or conjunctiva, the reaction of the pupil to light, nor with the normal consentaneous movements of winking. Its effects are, therefore, confined to the exercise of visual sensibility.

Christiani on the Influence of the Brain on Respiration, &c.—Christiani (*Acad. d. Wissensch. zu Berlin*, February, 1881, Sep. Abdruck) describes certain experiments relating to the influence of the brain and nerves on the respiratory mechanism; and also the effects of certain lesions on the animal's powers generally.

He finds that the inspiratory mechanism is stimulated by irritation, electrical, mechanical and otherwise, of certain sensory nerves, the optic, auditory, tactile, as well as by means of certain vagus fibres. Irritation also of a point in the wall of the third ventricle close to the corpora quadrigemina causes deep and quickened inspiratory movements. On the other hand irritation of the trigeminus, or of a point in the anterior tubercles of the corpora quadrigemina,

just under the aqueduct of Sylvius, causes expiratory movements. This action is associated with contraction of the pupil, whereas the inspiratory centre is associated with dilatation.

These "centres" are modified in their action by certain drugs, increased by strychnine, depressed by chloral.

Inspiratory action is also caused by irritation just posterior to the anterior tubercles of the corpora quadrigemina, confirmatory of previous researches on this subject.

Rabbits, after removal of the hemispheres and corpora striata, remain capable of locomotion, apparently even spontaneously. The action to sensory stimulation seems increased.

When section was made just anterior to the corpora quadrigemina, or injury made in the region of his "inspiratory centre" co-ordinated locomotion was no longer possible.

After the removal of the optic thalami and anterior surface of the corpora quadrigemina, reflex movements from mechanical stimulation of the hinder extremities were difficult to obtain, but irritation of the trigeminus or pinching the tail caused the utterance of peculiar cries. After section posterior to the corpora quadrigemina tetanic spasms occurred, and were readily excited by the slightest touch as in strychnine poisoning. This tetanic condition subsides, and leaves increased reflex excitability.

Animals possessing only the medulla, cerebellum and pons, make defensive movements if the nose or conjunctiva is irritated.

After destruction of the third ventricle or section posterior to the corpora quadrigemina, strychnine appears to act less energetically. Strychnine seems specially to act on the complex ganglion centres of the optic thalami.

Adamkiewicz on the Influence of Sinapisms on Sensibility.—Adamkiewicz (*Berlin. klin. Wochensch.* No. 12, 1881), in a lecture to the physicians of Cracow, relates some interesting observations on the influence of mustard plasters on anæsthesia and normal sensation. In previous researches he had shown that the secretion of sweat was dependent on a bilaterally functioning centre in the medulla oblongata, acting both through cerebro-spinal and sympathetic tracts. Taking up the subject of "transfer" of sensibility, so well known in connection with metallo-therapy, he found that sinapisms produced precisely the same effect as metals.

In a case of organic hemianæsthesia (proved by a post-mortem examination to be dependent on hæmorrhage into the lenticular nucleus and internal capsule), he succeeded by sinapisms applied to

the anæsthetic side, in restoring sensibility on the whole of the paralysed side, lasting several days. The restoration was not permanent. There was no transference.

In the various forms of hysterical anæsthesia the results were variable. In certain irregularly distributed anæsthesiæ, sinapisms restored sensibility for a greater or less duration. This is not in relation with the condition as to vascularity.

In a case of hysterical hemianæsthesia, sinapisms produced all the phenomena of transfer. After a few days the old condition returned, but on renewal of the sinapisms sensibility was completely restored without being lost on the other side.

In another case of hemianæsthesia and hemiparesis complete transfer occurred also with lowering of the temperature 5° on the sound side, and corresponding rise on the side formerly anæsthetic.

Following out indications supplied by these observations he investigated the influence of sinapisms in normal individuals and found that the sensibility for touch and pain increases on the side of irritation, and diminishes correspondingly on the symmetrically placed point on the other side. Hence it appears these forms of sensibility are bilaterally organised in the nerve centres, and functionally antagonistic, as shown by the diminution of the one when the other is increased. The result also seems to be that the sense of touch and pain is independent of the richness of the part in nerves, and is a purely qualitative function of the skin.

As regards the thermal sense, however, it was found that though it is also rendered finer by sinapisms at the point of irritation, yet it is not correspondingly diminished on the other side. The thermal sense, therefore, does not appear to be bilaterally organised.

In a short appendix to the paper, data are given showing that the diminution of sensibility on the side not stimulated by sinapisms is not related to the circulation, though irritation of the skin causes bilateral changes in the circulation just as it does in the case of sweat-secretion.

Grainger Stewart on Peripheral Paralysis of Hands and Feet.—Dr. Grainger Stewart (*Edin. Med. Journ.* April, 1881), describes three cases of the co-existence of sensory, motor and trophic disorders in the hands and feet, most marked in the distal points, and not corresponding to the areas of distribution of particular nerves.

In one of the cases, fatal from pneumonia, degeneration was found in the cervical and slightly in the lumbar enlargement,

affecting the columns of Goll and the direct cerebellar tracts of Flechsig. The grey matter was unaffected. The median, ulnar and tibial nerves also exhibited morbid appearances microscopically, the process consisting in a breaking up of the axis cylinder. The morbid process did not affect the large cords of the plexuses.

Dr. Stewart believes the degeneration commenced in the periphery of the nerves and spread upwards.

He gives references to similar cases recorded in various quarters, and indicates the points of difference between this affection and other forms of paralysis. Recovery is the rule, but it may in some cases lead to atrophic paralysis, or extend into the spinal cord and cause death by affecting vital centres.

James on Deafmutism and Dizziness.—Professor James (*Harvard University Bulletin*, April 1, 1881) publishes an interesting preliminary note of an investigation he has made as to the sense of dizziness in deaf mutes. He was led to the inquiry by the known relationship between the semicircular canals and the sense of rotation, and by the speculation that, therefore, deaf mutes would be deficient in this respect. The result of actual experiment was to confirm this in the most marked manner. A very large number of deaf mutes are either wholly incapable of being made dizzy by the most violent rotations, or experience but a slight and transient giddiness. Others are strongly and normally affected. The deficiency seems quite independent of the age at which the deafness begins, semi-mutes and congenitals being found indifferently in all classes. Disorders of locomotion are also common among deaf mutes, a subject which deserves careful attention from physiologists.

From his researches Dr. James expresses the opinion that the normal guiding sensation in locomotion is that from the semicircular canals. This is co-ordinated in the cerebellum with the appropriate muscles, and the mechanism becomes structurally organised in the first few years of life. If the guiding sensation is abolished by disease, compensation must be effected through sight and touch. This, however, is affected but slowly, and hence for many years the patient's gait is uncertain, especially in the dark. When the defect is congenital, the cerebellar mechanism is organised from the outset in co-ordination with tactile sensations, and no difficulty occurs.

An extended examination of typical cases is desirable, in order to test the value of this hypothesis.

Brachial Monoplegia (Traumatic).—Stimson (*Archives of Medicine*, April 1881) records an interesting case in which trephining was practised for a cerebral abscess resulting from traumatic lesion of the right side of the head above the ear, two months and a half previously. Besides the general indications of cerebral abscess, there were localising symptoms in the form of wrist-drop on the left side.

The Rolandic line was found to pass about half an inch in front of the upper end of the scar. A small perforation of the skull was found at this point, and on the trephine being applied just anterior to the opening, an abscess was found a quarter of an inch below the surface. About two ounces of pus were withdrawn after incision into the dura and brain substance. The patient died nine hours after the operation.

The seat of the abscess was at the point immediately posterior to the lower third of the ascending parietal convolution—the centres for the hand and wrist.

DAVID FERRIER.

Buzzard on the affection of Bones and Joints in Locomotor Ataxy, and its association with gastric crises. (*Transactions of the Pathological Society of London for* 1880; *British Medical Journal*, March 5th, 1881.)—Since 1868, when Professor Charcot first directed attention to the peculiar arthropathy associated with locomotor ataxy, the subject has engaged the attention of numerous observers abroad, especially in France. It is remarkable that in England there have been but few references to it. With the exception of a brief record of a case by Dr. Clifford Allbutt (*St. George's Hospital Reports*, 1869), Dr. Buzzard was the first to refer to it, in a clinical lecture upon a typical case of the kind, published in the *Lancet*, August 22, 1874. Since that time he has collected seven more cases, notes of which are published in the communications before us. The following is a brief description of these:—

1. A man with complete disorganisation of the right hip-joint, the head and neck of the femur having entirely disappeared in a few months. In this case a peculiar splint of bone had developed in the quadriceps femoris muscle.

2. A woman with spontaneous fracture of both femora.

3. A woman with disorganisation of both knee-joints, and incipient arthropathy of the right shoulder-joint.

4. A man with complete disorganisation of the left knee-joint.

5. A man with disorganisation of the left knee-joint.

6. A man in whom the head of the left humerus had disappeared.

7. A woman with arthropathy of the left hip-joint. The head of the femur had disappeared.

8. A man with arthropathy of the right knee-joint.

This is a simple list of the cases. Reference must be made to the papers for ample details, which it would occupy too much space to reproduce here. It is sufficient to say that the descriptions leave no doubt that they were all typical cases of tabes dorsalis.

A very interesting and original suggestion is made by Dr. Buzzard in reference to these cases. He was much struck by the fact that two of the female patients referred to above, who were under his care at the same time, suffered from the typical *crises gastriques* of Charcot. His experience told him that both the arthropathy and the gastric crises were very exceptional symptoms in tabes dorsalis. Their coincidence in two cases suggested to him the idea of inquiring whether there were grounds for supposing the association to be something more than a strange coincidence. The result of the inquiry is very remarkable. He has found that out of thirty cases of affections of bones and joints in tabes (which includes eight of his own) no fewer than fourteen, or nearly half, were affected with typical gastric crises. Whilst searching for data he came across a remark of Professor Ball in his paper on the subject (*Gazette des Hôpitaux*, 1868–69), in which he notes that in one-fourth of his cases "visceral troubles" were associated with the articular lesions.

Buzzard shows that the idea of Charcot that the articular lesions would be found to depend on lesion of the ganglionic cells in the anterior cornua, has not been supported by investigation, and he adduces numerous reasons which appear to prove conclusively that it must be abandoned. Starting with the suggestion that the gastric crises may be reasonably supposed to depend upon lesion of the roots of the vagi homologous with the lesion of the posterior columns of the cord, Buzzard suggests that the lesion of joints and bones probably depends upon invasion of a part of the medulla oblongata close to the roots of the vagi. He asks, Is there something which we may call provisionally a trophic centre for the osseous and articulatory system in the immediate neighbourhood of the roots of the vagi? The discovery of such a centre would materially help us, he adds, to explain the remarkable association of cardiac complications with the joint affection of acute rheumatism, as well as the sweating characteristic of this disease, and the

occasional hyper-pyrexia which occurs in it, and it might also help us to throw light upon the pathology of arthritis deformans.

It is interesting to note that after Dr. Buzzard had called attention at the Pathological Society to the frequency of association of gastric crises with the arthropathy of tabes dorsalis, the President (Mr. Hutchinson) related some particulars of a case which also showed this association; and Dr. Sturge referred to another, in which a similar association was to be found. When, later on, Buzzard was pursuing the subject, he met with four more cases of arthropathy, in three of which again typical gastric crises formed part of the history.

In a work by Dr. X. Arnozan, recently published, 'Des Lésions trophiques consécutives aux maladies du système nerveux,' exception is taken to Buzzard's hypothesis on the grounds that gastric crises are so common that the association is not important, and that there is no evidence that they are caused by sclerosis of the roots of the vagus. Arnozan thinks the seat of osseous lesion must be sought in the sensory regions of the cord.

Buzzard on Acute anterior Polio-myelitis in infants and adults. (*Lancet*, December 4 and 25, 1880; *Medical Press and Circular*, January 26, February 2, 9, 16, 23, March 2, 1881.)—In the course of two lectures, delivered at the National Hospital for the Paralysed and Epileptic, several patients were exhibited, and the symptoms, diagnosis, prognosis, etiology, and treatment of the disease were discussed. In reference to diagnosis, Buzzard points out that muscles in the district of a particular nerve are often found very unequally affected, some being completely paralysed, others but slightly affected. This, with the absence of cutaneous anæsthesia, points to a central origin, not an affection of a mixed spinal nerve-trunk. Where the quadriceps extensor femoris is paralysed there is absence of patellar tendon reflex. Buzzard has frequently observed that during regression many muscles recover their excitability to voluntary impulses long before they respond to the induced current, and he makes the following original suggestion as a possible explanation of this anomaly. He points out that according to Huguenin the anterior root is composed of fibres of which, although the greater part are connected with the ganglionic cells in the anterior horn, some ascend in the lateral column distinct from such connection. These fibres not having their trophic centre in the ganglionic cells, but higher up, would escape, he suggests, all but a temporary obstruction of function,

and would convey motor impulses to the muscles sufficient to cause movement. He lays stress on the condition of patellar tendon reflex as a convenient mode of diagnosis between this disease and cerebral paralysis. In the former it is absent, in the latter either unaffected or still more often in excess. Two patients were shown to illustrate this point: a hemiplegic form of infantile paralysis, and a case of cerebral hemiplegia in an infant.

Buzzard demonstrated the reaction of degeneration, and suggests that simply by using the letter Z (German Zückung) the English notation can be assimilated to the German, and comparison much facilitated. He uses K—kathode; A—anode; S (shutting—schlieszung); O (opening—oeffnung). He remarks that the Germans have set us an example by adopting Faraday's terms, Kathode and Anode.

He relates (in reference to diagnosis) the case of an infant who suddenly lost the use of a lower extremity after a little febrile disturbance. A large amount of uric acid was found in the urine, accounted for, he thinks, by the child having been nourished on a milk food containing an enormous quantity of sugar. Buzzard supposes that minute crystals of uric acid, or some equivalent, had got deposited in the loose connective tissue between the muscles (expansions of the lymphatic system), and set up some inflammation. The suggestion of this as a cause of symptoms he owed to Dr. Burdon-Sanderson.

A patient was shown with paralysis of the right arm, left leg, and *right side of the face*. The latter unusual complication of this disease he attributes to extension of the affection upwards to the nucleus of the portio dura, and suggests that the reason why we do not often witness the association is because the disease when it invades the bulb is likely to prove fatal by involving the roots of the vagus also. Cases of unexplained death may more often occur from this cause than is commonly supposed. Buzzard makes the important observation that such a thing as monoplegia is extremely rare in this disease. Limbs supposed to be unaffected are only comparatively so, as shown by lowered faradic excitability, and absence of tendon reflex.

He notes that alcohol must be absolved from the charge of causation. The patients most numerously affected are children. Some adult cases brought forward by him were abstainers.

The atrophy of the muscles is, for a long while at least, simple atrophy. It is the affection of the ganglionic cells, and nerve fibres proceeding from them, which is the serious lesion; and

Buzzard does not see much use in stimulating the *muscular* fibres by electrical currents. Indeed there is a disadvantage, at least in the early stage of the disease, for the stimulation of the skin being conveyed to the cord tends to irritate the ganglionic cells in the anterior horn.

Nor, when deformities are commencing, would he encourage voluntary movements of a limb, for by so doing overaction of the muscle least affected is encouraged, and the deformity is thereby increased. He enjoins rest in bed, ergot as early as possible, a mild constant current (stabile) to the spinal cord, and a light apparatus to check deformity as far as possible.

Buzzard on Tendon Reflex in the Diagnosis of Diseases of the Spinal Cord. (*Lancet*, November 27th and December 4th, 1880.)—In previous papers (*Lancet*, July 27th and August 4, 1878; *Brain*, July, 1878; *Lancet*, January 18, 1879, January 10, February 14, March 20, and April, 1880) Buzzard has described in detail the knee and foot phenomena of Westphal and Erb. The present paper is a contribution to the arguments which the clinical facts afford in favour of the reflex mechanism of the phenomena. Recognising that some eminent physiologists discard this view, principally on the ground that the interval of time between the blow and contraction is too short for a true reflex, Buzzard thinks that either there may be some source of fallacy in measurement, or that a shorter period of time may be required for conduction through the centre where, as in the present case, the centripetal stimulation is strong and rough. The absence of tendon reflex in tabes, along with preserved skin reflex, shows, he suggests, that the path of conduction must differ in the two phenomena, and that they cannot properly, therefore, be compared as regards time.

Buzzard points out that a convenient mode of testing the patellar tendon reflex is by lightly touching the patient's thigh with the left hand, whilst with the right a blow is given with a percussion stethoscope or hammer on the ligamentum patellæ, the patient being seated with his foot resting firmly upon the ground, and slightly advanced. The quadriceps muscle is felt to contract. The mode is especially convenient for the examination of females.

By a rough diagram Buzzard shows the nervous arc of communication extending from the tendon to the posterior roots, anterior horn, anterior roots, and thus to the muscle. He remarks that the integrity of this arc ensures the contraction of the muscle (if *it* be

healthy) to a blow on the tendon. On the other hand, the response to a blow *nearly always* proves that the arc is not seriously interrupted; but he makes the important observation that there is an exception to this rule. There may be atrophy of some ganglion cells in the anterior horn. If this lesion existed alone, tendon reflex (as shown by another diagram) would be abolished; but if the lateral column be sclerosed, the inhibitory influence from above is so much interrupted that reflex occurs. In cases, therefore, of lateral sclerosis with muscular atrophy, and also in cases of tabes dorsalis with lateral sclerosis, we may get the patellar tendon reflex preserved although the nervous arc is not intact. He ascribes the exaggeration of tendon reflex to the interception of controlling influence which comes down through the lateral column, and points out that the absence of this influence (e.g. in a case of hysterical paralysis) produces similar effects to its mechanical interruption by means of sclerosis, though in the former case they are only temporary. He points out that in spastic paraplegia, secondary to myelitis, the exaggeration of reflex may be connected with a temporary interruption (something short of sclerosis) in the lateral column, and may disappear as the lesion clears up.

Buzzard has in several cases witnessed the occurrence of ankle clonus in hysteria, which in one case ceased suddenly, so to speak, on the patient's recovery. He points out, therefore, that the occurrence of ankle clonus must not be regarded as conclusive evidence of a structural lesion of the spinal cord. Buzzard describes an extremely interesting case of a man in whom the patellar tendon reflex was absent on each side during an attack of syphilitic myelitis, which apparently affected a lateral half of the cord. In the limb corresponding to the seat of lesion, the left, it was absent in connection with motor paralysis and muscular wasting, the facts apparently pointing to the arc being "cut" in the anterior horn. In the right limb it was also absent, but in entirely different circumstances, for here the motor power was perfect, and the muscle not wasted, but the cutaneous sensibility was lost. To cause this affection of sensibility, the sensory part of this arc must have been cut. This showed that the lesion, although largely confined to the left half of the cord, encroached a little upon the posterior grey matter of the right half also. In confirmation of this view, Buzzard points to the fact that there was a patch of cutaneous anæsthesia in the neighbourhood of the left trochanter.

Buzzard discusses an interesting point, to which he was the first

to draw attention in 1878—the fact that in tabes dorsalis, with an entire absence of patellar tendon reflex, the muscle is often abnormally irritable to direct percussion. Erb has since (1879) recorded a similar observation. Buzzard finds an explanation of the circumstance in the irritative lesion of the posterior root fibres, and classes it along with the tendency to cramp in sciatica, and clonic spasm of facial muscles in tic douloureux. The case of a patient of his, in whom he had had the infra-orbital nerve stretched for neuralgia, suggested to Buzzard the ingenious and original idea of comparing the tendon reflex in a muscle on each side of the face. He points out that the fifth nerve practically represents the posterior root of a spinal nerve, of which the portio dura is the anterior root, and suggests that in the face we have an opportunity of observation to be found nowhere else, owing to the separation of roots, which in a spinal nerve are bound up in one trunk. He found that he could cause contraction of the zygomaticus major muscle by striking its tendinous origin on the malar bone. In the case in which the sensory root had been stretched (and therefore presumably to a certain extent physiologically impaired), the response on the side of lesion was distinctly less than that on the sound side, although an occasional clonic spasm of the muscles (which had long marked the case—one of severe tic)—seemed to show that the muscle itself was abnormally irritable. The case here appears to Buzzard to resemble strikingly what happens in tabes where tendon reflex is lost in connection with lesion of the sensory portion of the arc, the muscle itself being abnormally irritable. In lateral sclerosis, Buzzard remarks, the converse will often be found to obtain. The tendon reflex will be excessive, whilst the muscles will not respond to a blow, and will require a distinctly stronger faradic current than in health to cause their contraction.

By means of various diagrams, Buzzard suggests explanations of the condition of tendon reflex obtaining in infantile paralysis, protopathic muscular atrophy, lateral sclerosis, deuteropathic muscular atrophy, &c. He makes the important suggestion that the patellar tendon reflex is lost somewhat easily, and that it will often be found wanting when there is but a slight lowering of faradic excitability. Great care is therefore necessary in testing electrically to prevent the error of ascribing the break which he supposes to occur in the nervous arc to a lesion in its sensory instead of its motor portion.

J. Hughlings-Jackson.

Laffont on the Electrisation of the Vago-sympathetic Cord. (*Progrès Médical*, No. 12, 1881.)—The author gives the result of his experiments with reference to the question much discussed lately, whether the stimulation of the sympathetic increases the flow of saliva and the circulation of the labio-buccal region on the same side, owing to the vaso-dilating fibres it is supposed to contain. When on a dog the vago-sympathetic is divided and to the cephalic end a weak current applied, secretion of saliva occurs, and the mucous membrane becomes pale. The latter effect, according to Dastre and Morat, is due to a reflex action, through the sound vagus, upon the heart, or, perhaps, to the excitation of the depressor nerve. But manometric measurements show that there is no diminution in the blood pressure; nor is there diminution, but acceleration of the heart's beats. Hence the pallor observed is due to the direct stimulation of the sympathetic. The dilatation which occurs on the same side after the excitation is purely passive. During the excitation (with a weak current) no vagus-reflex is observed on the same side, but occurs on the opposite side, where the corresponding side is the seat of vaso-dilatation. With stronger currents dilatation appears on both sides, because the powerful vagus-reflex action overcomes the direct excitation of the sympathetic vaso-motors. That the sympathetic in the dog is a purely vaso-constrictor nerve was well shown in a case where the nerve was naturally distinct from the vagus: here no dilatation was observed. The flow of saliva in such experiments is not continuous as long as derived currents through the vagus are carefully excluded.

Rählmann on the Neuro-Pathological Significance of the Pupillary Dilatation. (*Volkmann's Sammlung*, No. 185.)

1. When on illumination the pupil of one eye does not contract, but does so consensually with the other, its optic nerve is still conducting, and there is either oculomotor paralysis, or the movements of the pupil are interfered with by atropia, iritis, atrophy, &c. Blindness of one eye is accompanied with sympathetic reaction when the pupil of the other is made to contract by light.

2. Complete blindness with pupils still reacting (as in uræmia) the lesion lies beyond the corpora quadrigemina.

3. If both pupils react during convergence, both oculomotors are conducting. In paralysis of the pupillary branch of these nerves there is dilatation and immobility, chiefly with complete oculomotor paresis. Unilateral paresis of the pupillary branch is probabably always syphilitic.

4. If both pupils react not to light but to convergence, and if there is some power of vision left, there is a lesion of the fibres of Meynert, between the corpora quadrigemina and the oculomotor nucleus. This condition often obtains in central disease.

5. The medium dilatation depends upon the intensity of the stimulus conveyed to the sympathetic elements of the cervical cord, from the sensory or physical excitations. In weak, nervous people, and in mania, the pupils are often wide. Contracted pupils in maniacal excitement are ominous of subsequent paralysis. Here also signs may occur of rhythmically interrupted action of the sympathetic: pupillary hippus.

6. Narrow pupils characterise all conditions, which depress the cortical functions, especially progressive paralysis of the insane.

7. Narrow pupils are peculiarly common spinal diseases (especially if cervical). In locomotor ataxy the reaction to light is absent, but is normal to convergence. Myosis in this disease is an early symptom.

8. Disturbed pupil diameter along with normal reactions depend upon sympathetic disturbances (megrim, saturnism, exophthalmic goitre, are characterised by irritation and consequent dilatation—contraction, with diminished lid aperture, indicate paresis or paralysis from injury, &c.).

9. Wide pupils are found in impeded respiration, carbonic acid poisoning (acting on the medullary sympathetic centres), Cheyne-Stokes respiration.

10. The same in brain pressure; hydrocephalus.

11. Irregularity shows disturbed sympathetic innervation. Unilateral dilatation with preserved movement is of grave cerebral import; (unilateral oculomotor paralysis is of no serious consequence). Alternating mydriasis, with preserved movement, yields a gloomy prognosis. In addition to this excessive susceptibility to atropia, and persistence of the phenomenon, along with perfect accommodation, are important differential factors. The condition here mentioned is frequent in mental disease and general paralysis.

Regis on La folie à deux. (*Gazette des Hôpitaux*, January 29, 1881.)—Dr. Régis, of the Asylum of St. Anne, recognises two varieties of this disease. First, that in which, of the two individuals who become insane, the one first attacked communicates his insanity to the second who remains on the borderlands of alienation, and may recover on being released from the dominating influence. Second, more complex and rare, where both are subject to the same form

of madness, having been exposed to the same morbid causes and predispositions.

The first division has been fully described by MM. Lasègue and Falret, and may be summarised in the following statements:—

Only one of the two is insane, he having communicated his delusions to the second by a certain moral and intellectual superiority. The individuals have lived in intimate contact. The active agent communicates part of his insanity to the passive; but between the two there is a line of demarcation, the first only being legally insane. Moreover, the latter speedily recovers when removed from the influence of the former.

The second division comprises those cases of true *folie à deux* presenting the following characteristics, as observed in five pairs of cases.

The two individuals become insane simultaneously. They are hereditarily predisposed to mental alienation. They live in intimate and constant contact. The exciting cause operates on both in the same way and at the same time. The mental manifestations resulting are substantially the same; the hallucinations and delusions are the same. Separation of the two is not attended by beneficial results.

Folie à deux may be defined as a monomania, usually of persecution, happening simultaneously to two individuals, the causes of which are—First, hereditary predisposition; Second, intimate and constant contact in daily life; Third, an exciting cause acting on both.

Bourneville and Regnard on Partial Epilepsy. (*Iconographie de la Salpétrière.* MM. Bourneville and Regnard.)—M. Charcot recognises three varieties of this affection: 1, partial epilepsy, with hemiplegia; 2, tonic partial epilepsy, or with contractions; 3, clonic partial epilepsy.

1. The first variety is composed of those who in infancy were seized by convulsions, followed by hemiplegia, and at a variable interval by epilepsy. This paralysis may persist, and a permanent hemiplegia result, or it may almost entirely disappear for a variable period, when it again returns and leaves an irremediable hemiplegia.

The access of this form of epilepsy is thus described. In general there is an absence of the initial outcry. *Tonic period.*—Rigidity of the paralysed side, or predominating in that side. *Clonic period.*—Clonic convulsions almost always limited to the paralysed

side; foaming at the mouth rare; still rarer involuntary micturition. *Period of stertor.*—Very brief, not profound, often absent. *Period of delirium.*—Quick return to consciousness, no epileptic delirium, the subsequent hebetude usually disappearing in a few weeks. After the fit there is a trembling of the paralysed members, and an elevation of the temperature as in ordinary epilepsy. There may be an isolated attack or a series of fits. In a certain number of cases they cease altogether with advancing years.

2. With regard to tonic partial epilepsy, the following are the principal symptoms as observed in one case. A little before the fit the patient complained of throbbing pain in the head, to which was soon added a feeling of constriction at the epigastrium and violent palpitation. In the fit the fingers stiffen and radiate, the face becomes pale, flashes of light pass before the eyes, the eyelids shut spasmodically, the muscles of the right side of the neck contract, there are convulsive movements of the eyelids, the jaws are strongly contracted. The right arm extended and pronated, the hand twisted on the arm rests on the back. The index and middle fingers are outstretched, the ring and little fingers are demi-flexed.

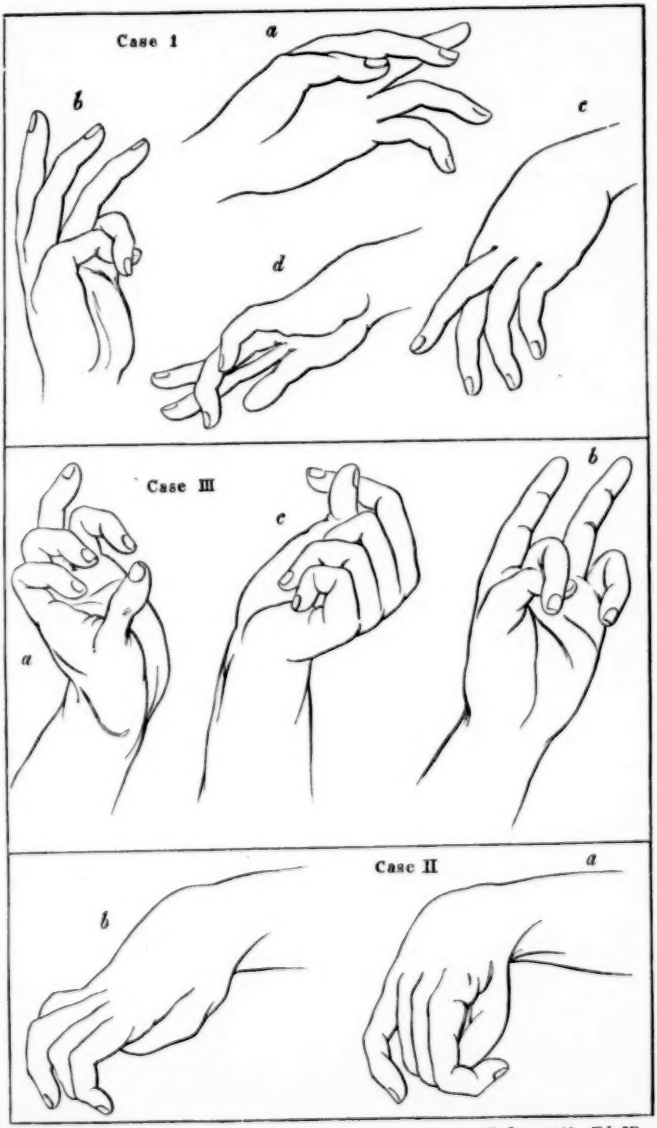
This condition lasts a minute, after which the patient rotates in his chair from left to right, the head being inclined to the shoulder. After two or three minutes these movements are reversed, and he progressively returns to his original position. There is no loss of consciousness.

3. In two cases of partial epilepsy, with clonic convulsions, the spasms were at first confined to a single limb; thence extended to the other limb of the same side, and finally to all four extremities. This form of epilepsy is more serious than the two preceding—one of the two cases having died, the other fallen into dementia.

Debove on a New Method of preparing the Spinal Cord for Microscopic Sections. (*Archives de Neurologie*, July, 1880.)—Dr. M. Debove recommends the following method of hardening the spinal cord for microscopic sections. Place the cord in a 4 per cent. solution of bichromate of ammonia for three weeks. Then in a solution of phenic gum for three days, and for three days more in alcohol. Sections may then be cut with the greatest facility, and placed in water to prevent their curling. They are then immersed in a saturated solution of picric acid for twenty-four hours, and coloured with carmine in twenty minutes—the picric acid acting as a mordant.

A. R. URQUHART, M.D.

CASES OF ATHETOSIS.



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